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METABOLISM OF PESTICIDES

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By

Calvin M. Menzie
Division of Pesticides Registration

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Bureau of Sport Fisheries and Wildlife
Special Scientific Report--Wildlife No. 127
Washington, D.C. · July 1969

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Acknowledgments

I should like to express my appreciation to Dr. Walter Benson of the Food and Drug Administration who took time to read portions of this manuscript and offered helpful comments and to Mrs. Phyllis Cook who typed this manuscript.

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METABOLISM OF PESTICIDES

Information concerning the fate of pesticides after application in the environment is useful to research workers who are concerned with their safe and effective use. It is important to know whether a compound will persist or whether it will be inactivated. Frequently, persistence is desirable for sustained control; for many uses, however, rapid detoxification of the compound may be desirable to avoid cumulative effects.

Disappearance of pesticides takes place by leaching, volatilization, adsorption, decomposition, or metabolism; and the use to which a particular pesticide is best suited may depend on the effects of one or more of these processes.

This review is primarily concerned with the metabolism and decomposition of pesticides. The importance of these processes is apparent upon consideration of the facts (1) that the duration of toxic action can be related to the rate and manner in which the compound is metabolized; (2) that the rate of elimination from the body is dependent upon the physico-chemical properties of the metabolic products; (3) that the ability of a compound to reach a site of action may be limited by the rate at which it is metabolized and the character of the metabolic products; and (4) that the toxicity of a compound can be decreased or intensified upon conversion to a metabolite. These points indicate the importance of knowing the pathways of metabolism as well as the degree of accumulation of metabolic products in tissue. From a more practical standpoint, the identification and measurement of residue levels of pesticides and their metabolites is necessary in order to fully evaluate the effects of pest control programs on wildlife.

Metabolic pathways can be classified into seven groups that cover the majority of biotransformations which pesticides undergo. These are:

1. Oxidation (in the sense that oxygen, as hydroxyl, takes part or is postulated to take part in one or more of the steps).
    - (a) Hydroxylation of aromatic rings
    - (b) Oxidation of side chains to alcohols, ketones, or carboxyl groups
    - (c) Dealkylation from oxygen or sulfur (ether cleavage)
    - (d) Sulfoxide formation
    - (e) N-oxide formation

2. Dehydrogenation and dehydrohalogenation

3. Reduction

4. Conjugation

    (a) Amide formation

    (b) Metal complex

    (c) Glucoside or Glucuronic acid

    (d) Sulfate

5. Hydrolytic Reactions

    (a) Cleavage of esters

    (b) Cleavage of amides

6. Exchange Reactions

7. Isomerization

Table I indicates the character of each of these reactions and gives illustrative examples of substances known to undergo each of the biotransformations indicated.

Table I

Some Metabolic Reactions Common to Pesticides

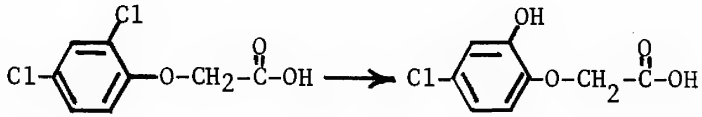
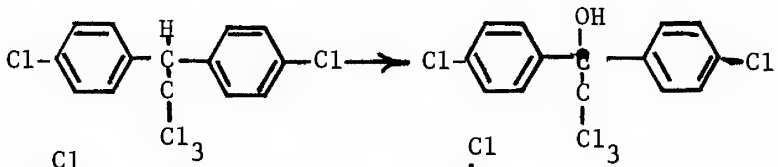
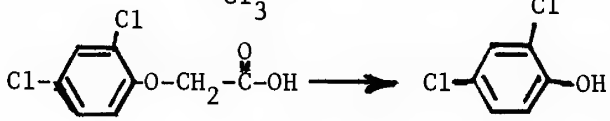
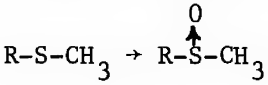
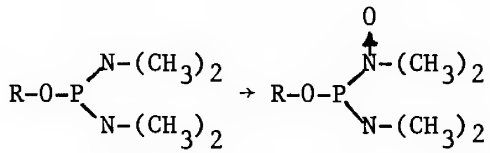
Pathway	Schematic	Pesticide which undergoes this reaction
1. Oxidation		
(a) Hydroxylation		2,4-D
(b) Side chain oxidation		DDT
(c) Ether cleavage		2,4-D
(d) Sulfoxide formation		Phorate
(e) N-oxide formation		Schraden

Table I (cont)

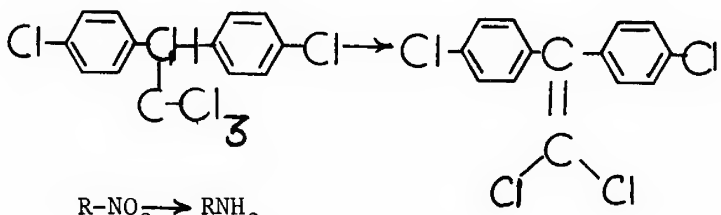
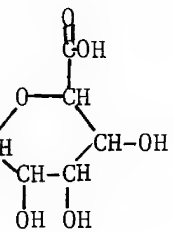
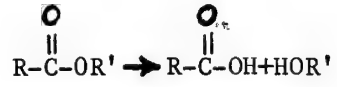
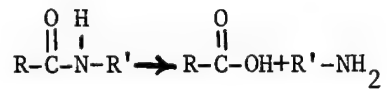
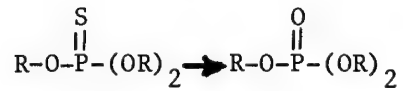
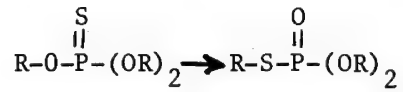
Pathway	Schematic	Pesticide which undergoes this reaction
2. Dehydrogenation and dehydrohalogenation		DDT
3. Reduction	$R\text{-}NO_2 \rightarrow RNH_2$	DNOC
4. Conjugation		
(a) Amide formation	$R\text{-}NH_2 + R'\text{-}COOH \rightarrow R\text{-}\overset{H}{\underset{|}{N}}\text{-}\overset{O}{\overset{\|\|}{C}}\text{-}R$	Amitrole
(b) Metal complex	$R_2\text{-}N + MeX \rightarrow (R_2N\text{-}Me)X$	Amitrole
(c) Glucoside and Glucuronic Acid	R-OH + Glucuronic Acid → 	Barthrin
(d) Sulfate	$ROH + SO_4^{=} \rightarrow R\text{-}O\text{-}\overset{O}{\overset{\|\|}{S}}\text{-}O^{-}$ $\quad\quad\quad

Table I (cont)

Pathway	Schematic	Pesticide which undergoes this reaction
5. Hydrolytic		
(a) Cleavage of esters		Malathion
(b) Cleavage of amides		Dimethoate
6. Exchange Reactions		Parathion
7. Isomerization		Most Organophosphates

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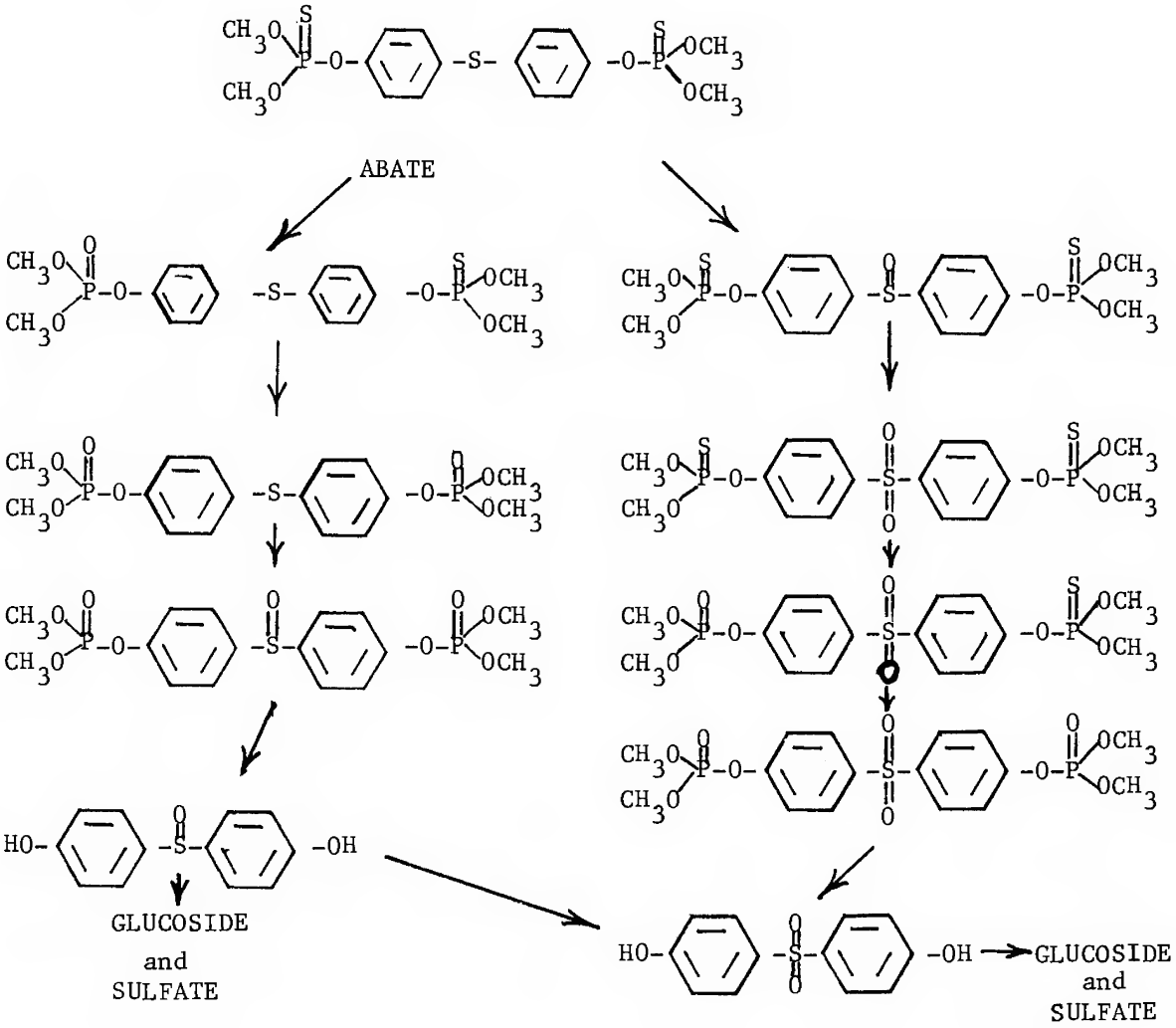
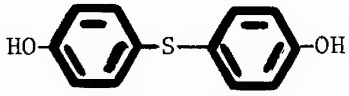
Abate (0,0,0',0'-tetramethyl 0,0'-thiodi-p-phenylene phosphorothioate)

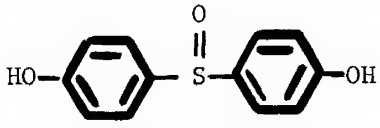
Studies with tritium-labeled Abate indicated that this insecticide was relatively resistant to metabolic degradation. Residues on bean leaves (Sieva lima bean) consisted primarily of intact Abate-about 70% of the applied dose. The major metabolite was the sulfoxide derivative-less than 5% of the dose. Traces of the sulfone derivative and the oxygen analog and glucosidic conjugates of the phenolic hydrolysis products from Abate and its sulfoxide and sulfone derivatives were also observed (127, 1553).



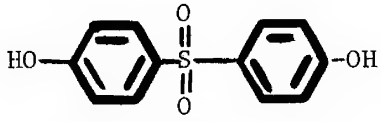
When rats were fed Abate, 60% of the material appeared in the feces as the oxygen analog of Abate and its sulfoxide. The thiodiphenol, sulfinyldiphenol and sulfonyldiphenol were also found. In the urine, sulfate and glucoside conjugates of the hydrolysis products of Abate and sulfoxide and sulfone analogs accounted for 39.5% of the material administered. All three hydrolysis products were also found in the free form. Only Abate and its sulfoxide were found in the fatty tissue. Five compounds found in feces and three in urine were not identified (1640, 1733).



Thiodiphenol



Sulfinyldiphenol



Sulfonyldiphenol

Acarol (Isopropyl bromobenzilate) [Isopropyl 2,2-bis(p-bromophenyl glycolate]

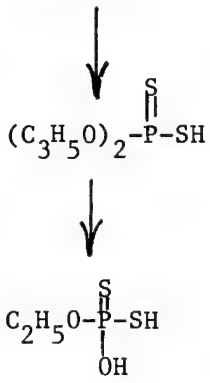
In soil, this material was converted to the corresponding phenone (1642).

Acethion (0,0-diethyl S-carboethoxymethylphosphorodithioate)

Degradation of acethion was rapid and 77-88% of the injected dose appeared as chloroform extractables within 0.5 hr. after treatment (1122). Chromatographic separation and analysis showed the principal metabolite to be acethion acid. Some mono- and diethyl phosphorodithioic acids were also found (840, 1099).

Acethion

Acethion Acid



Aldrin was readily converted to dieldrin in the body of beef and dairy cattle, pigs, sheep, rats, and poultry (79). This change was independent of the mode of entry, since it occurred following oral ingestion or subcutaneous injection.

When aldrin ^{-14}C was fed to male rats (847, 903), or orally injected into male and female rats (640), the active material excreted in feces and urine consisted of aldrin, dieldrin, and considerable amounts (up to 75% in feces and up to 95% in urine), of a mixture of unidentified hydrophilic metabolites. At a feeding level of 4.3 mcg per day, a saturation level was reached after about eight weeks. Daily excretion of active material approximated the entire activity administered daily. If injected, 16.2% of aldrin-C^{14} and 13% of dieldrin-C^{14} was excreted within four hours, via the bile into the intestinal canal, mainly as hydrophylic compounds (830, 1041). Recent studies with rat and rabbit livers showed that this epoxidation was performed by the microsomes (1069, 1521).

Paper chromatography of extracts of feces and urine showed a high percentage of aldrin-C^{14} initially. The percentage of unchanged aldrin then decreased while that of hydrophylic metabolites increased continuously for about twelve days; and the distribution of excreted compounds remained unchanged as long as aldrin was administered daily. After aldrin administration was discontinued, the relative percentage of aldrin in feces decreased and dieldrin increased (847, 903). Paper and thin-layer chromatography showed that two different compounds were in the feces; that two different compounds were in the urine; and that one of the compounds in feces behaved like one of those in the urine. Hydrolysis of the main compound from urine with alcoholic KOH gave a new acidic compound (903). When rabbits were given aldrin intravenously, hydrolysis of the metabolites gave the aldrin diol (830).

Dieldrin was administered internally to rats as a single dose of 20 mg/kg body weight. During the first 8 days, unchanged dieldrin was excreted in urine. Within the following 4 days, a product of dieldrin was excreted. Of the dieldrin administered, 15.7% was excreted within 14 days in the feces (1253). Other feeding studies with rats have also shown that dieldrin is metabolized to two compounds more polar than dieldrin and unstable to alcoholic KOH (338). On the basis of elemental analysis, mass spectra, I.R. and Raman spectra, the structure of compound XI was proposed (335).

When labeled dieldrin was administered to a rabbit via stomach tube, six metabolites were isolated and purified via thin-layer chromatography. The main metabolite was identified as one of the two enantiomorphic isomers of 6,7-trans dihydroxy-dihydro-aldrin (aldrin diol), with a specific rotation of -13.7. Identification was confirmed by gas chromatography. After intravenous administration of this compound in rats, a more hydrophylic compound was found which corresponded to one of the other five compounds after

dieldrin administration. It was postulated that a carbonyl group was present (826, 902).

Studies with C^{14} labeled aldrin and dieldrin showed that, after intravenous injection in rats, radioactivity increased in the order of liver, duodenum, intestine, and feces and that the percentage of metabolites increased in the same fashion (827). In male rabbits, the major portion of the radioactivity was excreted in the urine, rather than the feces, after aldrin-C^{14} injection. Using column and thin-layer chromatography, eight metabolites were obtained from urine. Metabolite II was observed only in the first 3 to 4 weeks. The main metabolite III (43%) was crystallized from n-hexane, m.p. 131.5°C, and was shown by mass spectroscopy to have a molecular weight of 399 and to contain 6 chlorine atoms. Microanalyses gave a formula of $C_{12}H_{10}O_2Cl_6$ and infra-red showed that the only functional groups were hydroxy. Synthesis showed this to be the optically active trans-6,7-aldrin-diol. Metabolite IV, found in small quantities (3%), was thought to be an isomer of III. Mass spectroscopy gave a M.W. of 399 and analytical results compatible with the structure of an aldrin-diol. The m.p. was 212°C. The infra-red spectrum was characteristic of an OH band at 3350 A. and the characteristic absorption band (at 1600A.) for the double bond of chlorinated rings was absent. Metabolite IV was not identical with cis-6,7-aldrin-diol (m.p. 217°C), prepared by $KMnO_4$ oxidation of aldrin. The aldrin-diol structure of metabolite IV was not further identified.

Metabolite VI (14%) was crystallized and the m.p. was 161° C. Hydrolysis with MeOH-KOH gave metabolite III. From mass spectroscopy, the M.W. was 483. Interpretation of I.R., mass spectra, and chemical analyses indicated a diacetyl of trans-6,7-aldrin-diol. The infra-red spectrum, analyses, and m.p. of the synthetic product and metabolite VI agreed.

Metabolite V had a dark yellow color, was resinous and could not be crystallized. After hydrolysis, metabolite III was demonstrated by T.L.C. Infra-red spectra were characteristic of the hydroxy band at 3570 A and a sharp C=O band at 1730 A. The acetate of trans-6,7-aldrin-diol agreed with the I.R. spectra but not with other analytical results of metabolite V.

Metabolite IIa was crystallized with m.p. 138-140° C. Analytical results agreed with that for dieldrin. The infra-red spectrum showed a C=O band at 1765 A. Metabolite IIa was apparently a ring ketone of undetermined structure. In untreated urine extracts, this compound could not be demonstrated by T.L.C. It was felt that this might be an artifact. Metabolite II was demonstrated by I.R. spectrum, analytical results and R_f values to be dieldrin (828).

In other studies, dieldrin was fed in a diet to mature male Carworth Farm strain rats for 6 months. Feces and urine were then collected separately for one month. Mass spectroscopy indicated that the urinary

metabolite had a M.W. = 358 and contained 5 chlorine atoms. Infrared spectra indicated the presence of an epoxide and a carbonyl and the absence of Cl-C=C-Cl. The compound formed a 2,4-dinitrophenylhydrazone. The results lead to the molecular formula of $C_{12}H_7Cl_5O_2$ and the structure 1,9,10,10,11-exo-pentachloro-4,5-epoxy-pentacyclo-(7,3,0,0,^{2,6},0^{3,8},0^{7,11}) dodecan-12-one for the urinary metabolite (XI).

The M.W. of the fecal metabolite was 394 and contained 6 chlorine atoms. Infrared spectra of the fecal metabolite indicated Cl-C=C-Cl, and $-CH_2-$, tertiary hydroxy, and an epoxide. This plus mass spectra lead to the empirical formula $C_{12}H_8Cl_6O_2$ and the structure 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-4a(or 5)-hydroxy-exo-1,4-endo-5,8-methanonaphalene (XII) (1591, 1658).

Regression lines for the relationship between concentration of dieldrin in tissues of pigeons was determined and an equation derived showing the change of concentration in the whole pigeon when exposure ceases.

$$(1)\quad C_t = C_o e^{-bt}$$

where b=0.0064

C_o =Concentration of dieldrin when exposure ceases.

From this relationship, the half-life of dieldrin in the pigeon has been calculated.

$$(2)\quad t_{1/2} = \frac{\log_{10} 2}{b} = 47.2 \text{ days } (P=0.95, 41.4 - 54.9)$$

An elimination mechanism of the type indicated by the equations implies that continuous uniform exposure will result in an equilibrium concentration in tissues expressed by equation (3).

$$(3)\quad C_t = C_{eq}\ (1-e^{bt}) \text{ where}$$

C_t = concentration at time t

C_{eq} = the asymptote ($C_t \rightarrow C_{eq}$ as $t \rightarrow \infty$

b = the slope of regression line when concentrations are transformed to corresponding logarithms.

(1225)

Urine of men with occupational exposure to dieldrin gave evidence that dieldrin was eliminated in humans as at least two neutral, polar, chlorinated metabolites, thus far unidentified (320, 464).

Application of aldrin to the American cockroach resulted in partial conversion to dieldrin (547). Similarly, tests with locusts (292) houseflies and mosquito (Aedes Aegypti) larvae (152, 829, 831, 536, 1155, 1428) showed that labeled aldrin was converted to dieldrin and hydrophylic compounds. In Musca vicina, studies demonstrated that the microsomes contained a system that was capable of causing this conversion and required NADPH and O_2 (1197). The sulfur and bromine analogs of dieldrin (S^{35} and Br^{82} labeled, respectively), also were shown to be partly metabolized to water-soluble and insoluble compounds (1514). With nerve components, studies have shown that dieldrin forms a complex that is inextractable with organic solvents (939). Culex pipiens quinquelfasciatus mosquitoes metabolized dieldrin to a compound believed to be the aldrin glycol (diol) (1113, 1625).

Housefly and pig liver microsomes hydroxylated dihydroaldrin, in the presence of NADPH, to a mixture of exo- and endo-6-hydroxydihydroaldrin (188, 1775).

The conversion of aldrin to dieldrin has been found to occur in soil, (138, 521, 523, 786, 829, 869, 870, 1500) in general. In 1956 it was found that carrots grown in aldrin-treated beds contained dieldrin (548). Similarly, peanuts grown in an aldrin-treated bed contained dieldrin residues in the hulls, meat, and vine forage (97). Aldrin was also converted to dieldrin in mushroom compost (1592), radishes and other plants (1783).

Ninety-two pure cultures were screened for aldrin-degrading activity. Most showed some capacity for converting aldrin to dieldrin. Of considerable interest was the ability of some fungi, actinomycetes, and bacteria to degrade dieldrin: trichoderms sp., fusarium, aspergillus, streptomyces, micromonospora, bacillus, nocardia and thermoactinomyces (1428).

In studies designed to discover microorganisms that could degrade dieldrin, ten such microbial cultures were obtained. Two were identified to species level as Trichoderma viride; six were found to belong to the genus Pseudomonas; and two, to the genus Bacillus. Using radioautography and thin-layer chromatography, in addition to dieldrin, nine compounds were indicated. One spot matched authentic 6,7-transdihydroxydihydroaldrin. The other spots were unidentified (936, 1599). In other studies, incubation with a pseudomonas sp. gave rise to nine compounds. Five of these were indicated: IV, XIII, XIV, XV, XVI (1684). Pseudomona melophthora degraded dieldrin, but no metabolites were identified (145). Trichoderma viride converted dieldrin into four metabolites, one of which was identified as 6,7-transdihydroxydihydroaldrin (1622).

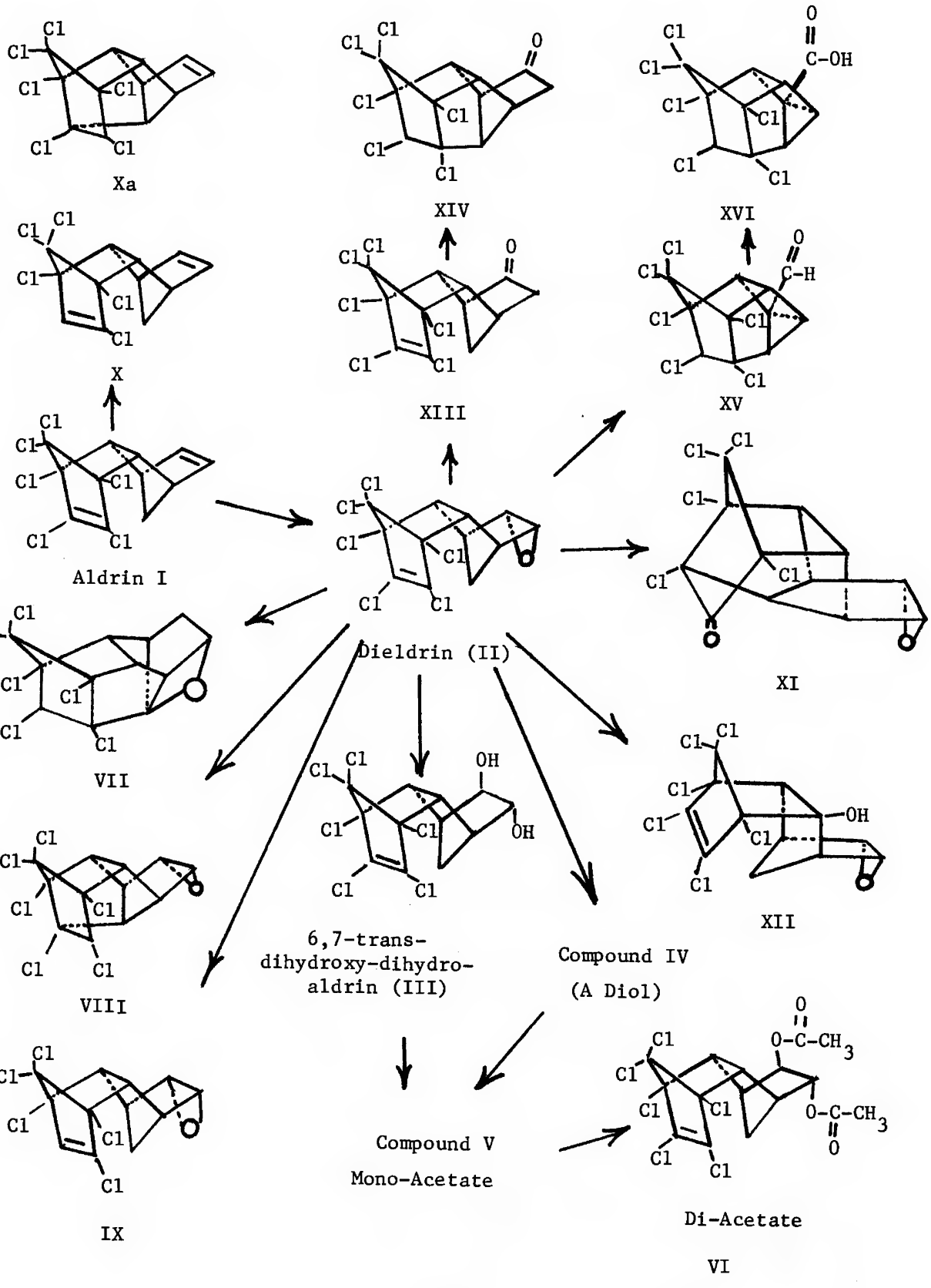
Aldrin was sprayed on apple trees in June and the rate of loss was observed. Twenty-four percent remained after one week and 2% after seven weeks. After eleven weeks, the residue consisted almost entirely of dieldrin and its UV irradiation products. By applying dieldrin at high rates, four conversion products were detected. Three of these were identical

on G.L.C. with compounds produced by UV irradiation of pure dieldrin in the laboratory. The main dieldrin irradiation product, prepared in the laboratory, had a m.p. 195-196°C and corresponded on G.L.C., T.L.C., and I.R. with that obtained in field trials. U.V. spectra showed the absence of a conjugated chromophore or a carbonyl group. I.R. spectra showed no absorption around 1600 cm^{-1}, the region ascribed to -CCl = CCl- bonding in spectra of dieldrin, aldrin, and heptachlor. There was no absorption corresponding to carbonyl (1540-1900 cm^{-1}) or hydroxyl (360 cm^{-1}) groups. The most prominent new peak at 870 cm^{-1}) was consistent with an oxygen atom attached to a tertiary carbon atom. The peak at 990 cm^{-1} corresponded with the absorption band of a 4-membered ring containing oxygen; and the two peaks at 1030 and 1070 cm^{-1} corresponded to absorption bands of 5-membered rings containing oxygen. NMR spectrometry showed the absence of the methano bridge present in the non-chlorinated ring in dieldrin. However, the M.W. and mass spectrum are the same as for dieldrin. One of the several isomeric forms (VII) was proposed (618).

Exposure of aldrin to sunlight and UV light produced two decomposition products which were isolated but not identified. Recrystallization from benzene-methanol gave crystalline compounds with m.p. of 178-9°C and 187-9°C (771). In the absence of air, ultra-violet irradiation of aldrin gave rise to two compounds whose suggested structures are those of compounds X and Xa. When aldrin was exposed to γ-radiation, dieldrin plus about eleven other compounds were observed (1678).

In other studies, gas chromatograms of herbage treated with dieldrin displayed an unknown peak in addition to dieldrin. UV irradiation of dieldrin at 2537 A gave rise to the same compound (1228). Subsequently, on the basis of I.R. spectra, G.L.C. and T.L.C. behavior, mass spectra, NMR spectra, molecular weight and elemental analyses, this ultraviolet irradiation product was shown to be 10-oxa-3,6-exo-4,5,13, 13-hexachlorohexacyclo ($6.3.1.1^{3,6}.1^{9,11}.0^{2,7}.0^{5,12}$) tridecane (VIII), m.p. 188°C. (1226, 1227, 1237, 1570).

Hexane solutions of dieldrin and aldrin were irradiated with UV light having a peak intensity of 2537 A. Elemental analysis indicated an empirical formula of $C_{12}H_9Cl_5O$ and $C_{12}H_9Cl_5$ for the respective photo-products, suggesting that dieldrin and aldrin had undergone monodechlorination during photolysis. Because of the similarity in chemical activity between the photo-products and dieldrin and aldrin, principal reliance was placed on spectrophotometric procedures for structure elucidation. On the basis of I.R., mass and NMR spectra, the photodecomposition product of dieldrin was assigned structure (IX) and that of aldrin was assigned structure (X) (645). High concentrations, in solution, or in the solid state, apparently favored the intramolecular reaction of carbon 2 with the adjacent hydrogen on the methylene bridge. In dilute solutions, with very large molar excess of hydrogen donor such as hexane, the replacement of chlorine by hydrogen appeared to predominate. From flies (Musca domestica L.) treated with photodieldrin(VIII) or photoaldrin (Xa), compound XI was obtained (1790).


Xa
XIV
XVI
X
XIII
XV
Aldrin I
Dieldrin (II)
XI
VII
6,7-trans-
dihydroxy-dihydro-
aldrin (III)
XII
VIII
Compound IV
(A Diol)
Compound V
Mono-Acetate
Di-Acetate
IX
VI

No metabolites could be demonstrated after exposure of daphnia and fat head minnows to endrin (1180). In the presence of $NADPH_2$, rat and rabbit liver microsomes converted isodrin to endrin (1069).

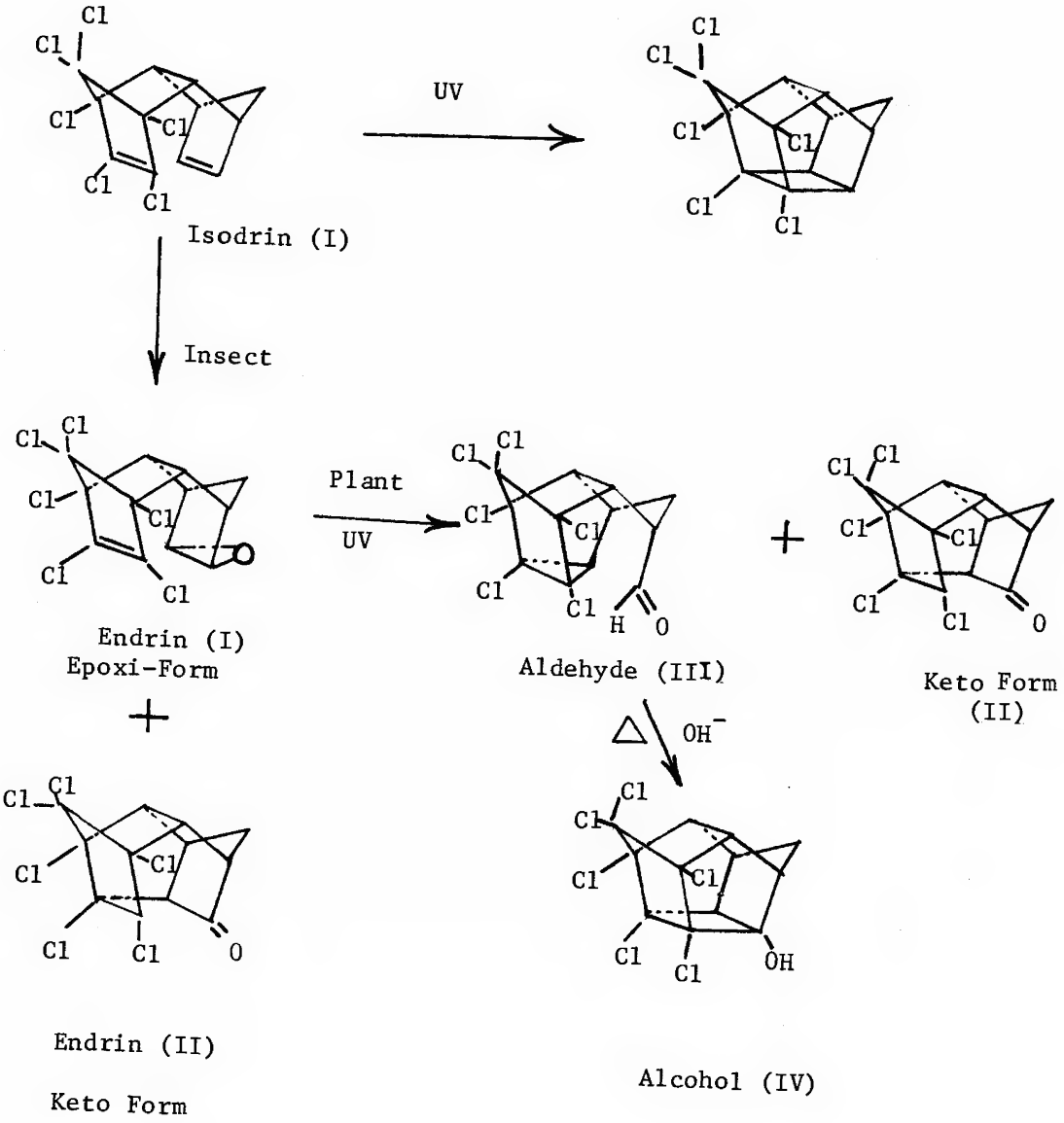
C^{14}-labeled endrin was administered orally and intravenously to rats. Examination of collected urine indicated a major metabolite more hydrophyllic than endrin--identified as the keto analog of endrin--and other hydrophyllic compound in lesser (about 5%) amount. These metabolites were identical with the $NADPH_2$-liver homogenate metabolite (1069, 1575).

On foliage endrin was less persistent than dieldrin. After one week, a small amount of the ketone isomer (II) of endrin was detected. This was not observed in later samples. The corresponding aldehyde (III) was present to the extent of 15% of the total residue after seven weeks but was not detected four weeks later (618). After application of endrin to Brassica oleracea var. capitata, two metabolites were observed. One was identified as the endrin-ketone. This occurred in darkness or in the presence of light and with plant homogenates as well as the intact plant (1600).

When isodrin ^{14}C was applied topically to houseflies, the corresponding epoxide endrin and small amounts of a water-insoluble product which behaved as the ketone, a known rearrangement product of endrin, were recovered (186). Endrin was not formed in tissues of heat-killed insects. Similarly, isodrin was converted to endrin by the American cockroach (Periplaneta americana L.) (546). Similar metabolic products were observed when Aspergillus flavus or mosquito larvae of Aedes aegypti were exposed to endrin (1575). Aerobacter aerogenes, Pseudomonas aeruginosa, and three unidentified marine organisms degraded endrin but no metabolites were identified (1561).

UV irradiation of endrin gave rise to one main compound and smaller amounts of several others (1228). After isolation of two irradiation products, infra-red spectra showed these to be identical with ketone (II) [1,8- exo-9,10,11,11-hexachloropentacyclo-(6.2.1.1.$^{3.6}$.$0^{2,7}0^{4,10}$.)dodecan-5-one] and aldehyde (III) [4,5,6,7,8,8-hexachlorohexahydro-4,7-methano-3,5,6-metheno-indan-1-carboxaldehyde] isomers of endrin (1237).

Endrin underwent thermal decomposition and isomerization beginning at about 90° to 100° C. Gas chromatography indicated about four compounds. By means of infra-red, one was identified as the ketone (II), 280-283° C (dec.); a second compound was the "cage" alcohol (IV), m.p. 330° C. An aldehyde (III) isomeric to (II) was also observed and identified after gas chromatography, infra-red, and elemental analysis (1159, 1160). Some spontaneous decomposition has also been observed (83).


UV
Isodrin (I)
Insect
Plant
UV
Endrin (I)
Epoxi-Form
Aldehyde (III)
Keto Form
(II)
OH^-
Endrin (II)
Keto Form
Alcohol (IV)

Studies have suggested that metabolic pathways for pyrethrins and allethrins differ and also that detoxification mechanisms in houseflies and roaches differ.

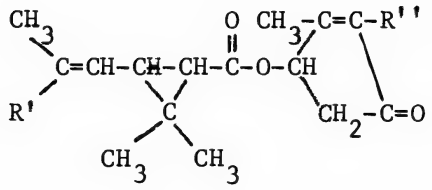
Metabolism of allethrin-C^{14} by a housefly enzyme system gave rise to 13 metabolites and each had the ester group intact. One compound represented over half the total radioactivity. Saponification of the major metabolite yielded a compound identified as chrysanthemumdicarboxylic acid. Chemical, I.R., and chromatographic analyses identified the major metabolite as 0-demethyl allethrin [3-(3-allyl-2-methyl-4-oxo-2-cyclopentenyl) chrysanthemumdicarboxylate] (1525). Dimethrin and Phthalthrin were also oxidized to their respective 0-demethyl pyrethrin II analogs.

Initially, it was suggested that decomposition of pyrethrins was caused by hydrolytic enzymes. Subsequent studies have shown that lipases of roaches and houseflies readily hydrolyze pyrethrin esters to keto alcohols, chrysanthemum acids, and several unidentified compounds (260, 1510, 1520, 1540). Some 8-12% of labeled pyrethrin applied to roaches was excreted as $C^{14}O_2$. When applied to houseflies, no $C^{14}O_2$ was detected (268, 690, 1515). Chrysanthemum acid and five unknown metabolites were detected. Three of the unknown metabolites had intact chrysanthemum acid moiety and an ester linkage. Cinerin I (268) behaved similarly but differed quantitatively. Thus, it appears that initial degradation occurred on the pyrethrolone or cinerolone moiety.

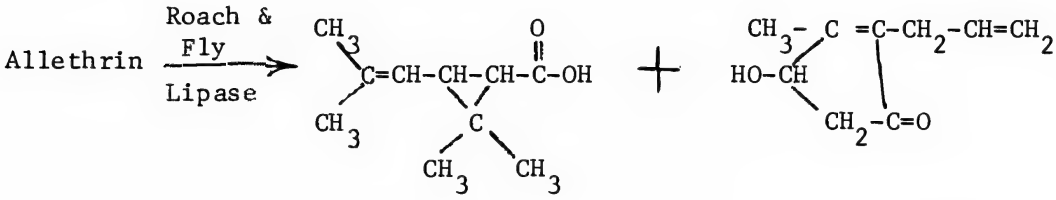
In other studies, pyrethrin I-C^{14} was converted *in vitro* by the housefly abdomen-$NADPH_2$ system to at least 10 metabolites. Saponification of the major metabolite gave rise to chrysanthemumdicarboxylic acid. Acid labeled dimethrin and phthalthrin gave 11 and 12 metabolites, respectively, and phthalthrin-N-methylene-C^{14} yielded 10 metabolites identical with those from acid labeling. Two compounds were detected only with acid labeling-hydrolyses products. The major metabolites were in each case identical with that from pyrethrin and allethrin and chemical studies indicated that the major metabolite of pyrethrin I, dimethrin, and phthalthrin were formed by oxidation of the analogous methyl group for allethrin (1525, 1526).

Metabolism of allethrin by *Musca domestica* L. was demonstrated. Metabolites were resolved chromatographically but not identified (174).

When pyrethrin I was administered as an anthelmintic, color tests on urine of humans indicated the presence of chrysanthemum monocarboxylic acid (43).



	R'	R''
Pyrethrin I	$-CH_3$	$-CH_2-CH=CH-CH=CH_2$
Pyrethrin II	$-\overset{O}{\overset{\|}{C}}-OCH_3$	$-CH_2-CH=CH-CH=CH_2$
Cinerin I	$-CH_3$	$-CH_2-CH=CH-CH_3$
Cinerin II	$-\overset{O}{\overset{\|}{C}}-OCH_3$	$-CH_2-CH=CH-CH_3$
Isopyrethrin I	$-CH_3$	$-CH=CH-CH=CH-CH_3$
Isopyrethrin II	$-\overset{O}{\overset{\|}{C}}-OCH_3$	$-CH=CH-CH=CH-CH_3$
Allethrin I	$-CH_3$	$-CH_2-CH=CH_2$
Allethrin II	$-C(=O)-OCH_3$	$-CH_2-CH=CH_2$



Chrysanthemum Monocarboxylic Acid

Allethrolone

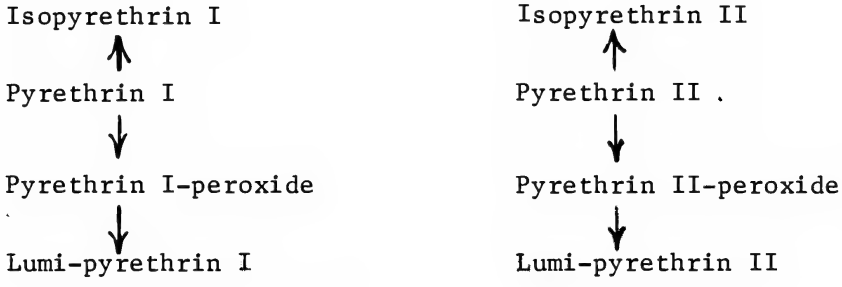
Studies with rats suggested that barthrin (6-chloropiperonyl chrysanthemumate) was metabolized and excreted in urine as metabolites, free and conjugated, having a strong reducing effect on Benedict's reagent (19, 930). Residues from urine gave a positive test for chrysanthemumic acid, for methylene oxide linkage, and for glucuronic acid before and after hydrolysis. Tentatively, it was concluded that barthrin in rats was metabolized and excreted in the urine as chrysanthemumic acid 6-chloropiperonlylic acid, and partly as the 6-chloropiperonyl glucuronide or glycine conjugate. Similar results were obtained with Dimethrin (2,4-Dimethylbenzyl chrysanthemumate) (20, 930).

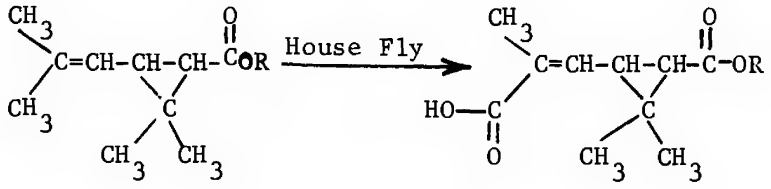
Male Wistar rats were administered phthalthrin orally and intravenously. Tissues, blood, feces and urine were processed and thin-layer and column chromatography were used to separate the metabolites. Elementary analyses, NMR, UV, and IR were used to identify the metabolites. Although residues were observed in tissues, only Cyclohexane-1,2-dicarboximide in liver was identified. From urine, N-Hydroxymethyl-3,4,5,6-Tetrahydrophthalimide, Cyclohexane-1,2-dicarboximide, 3-or-4-hydroxycyclohexane-1,2-dicarboximide and its glucuronide were obtained and identified. The major metabolite from in vivo and in vitro (liver homogenate) studies was N-Hydroxymethyl-3,4,5,6-Tetrahydrophthalimide (1584).

The synergist piperonyl cyclonene inhibited the detoxification of pyrethrin more than that of allethrin. C^{-14} labeled allethrin was metabolized by flies in large part to a polar compound with the same R_f as allethrolone (690).

Allethrin exhibited more stability towards ultra-violet light than did the natural pyrethrins (119). Studies showed that pyrethrum was sensitive to ultra-violet in the range of 2200-2300 Angstroms (1054) and underwent some decomposition. This effect was independent of oxygen or humidity (117, 118).

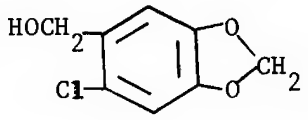
In light and air, pyrethrin I was inactivated by conversion to lumi-pyrethrin I via pyrethrin I-peroxide. Pyrethrin II was similarly affected. Under the influence of heat, pyrethrin I and II were converted to their respective isopyrethrins. These were affected by light and air in the same manner as pyrethrin I and II (1345).



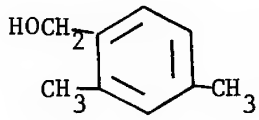


R

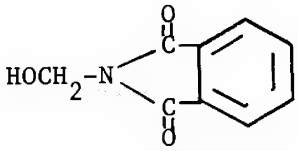
Barthrin

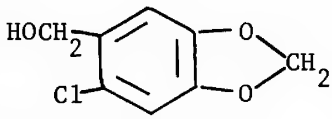


Dimethrin

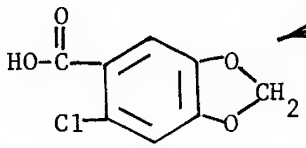


Phthalthrin

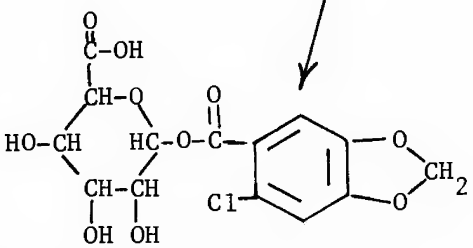


Barthrin —Rat→ Chrysanthemum monocarboxylic Acid + 

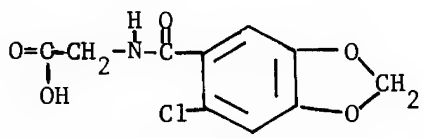
6-Chloropiperonyl Alcohol



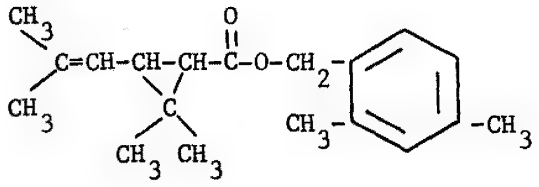
6-Chloropiperonylic Acid



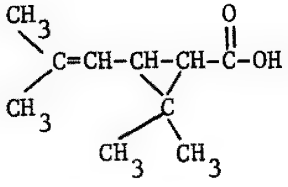
6-Chloropiperonyl Glucuronide



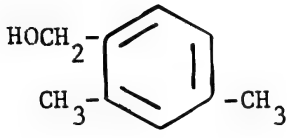
6-Chloropiperonuric Acid



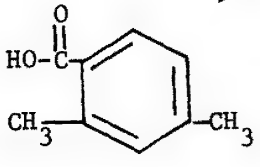
Dimethrin



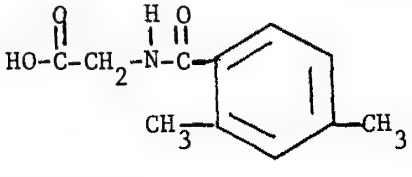
Chrysanthemum
Monocarboxylic Acid



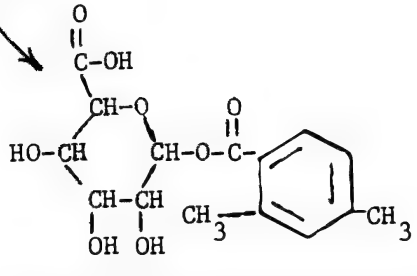
2,4-Dimethyl
Benzyl Alcohol



2,4-Dimethyl Benzoic Acid
(2,4-Xylic Acid)



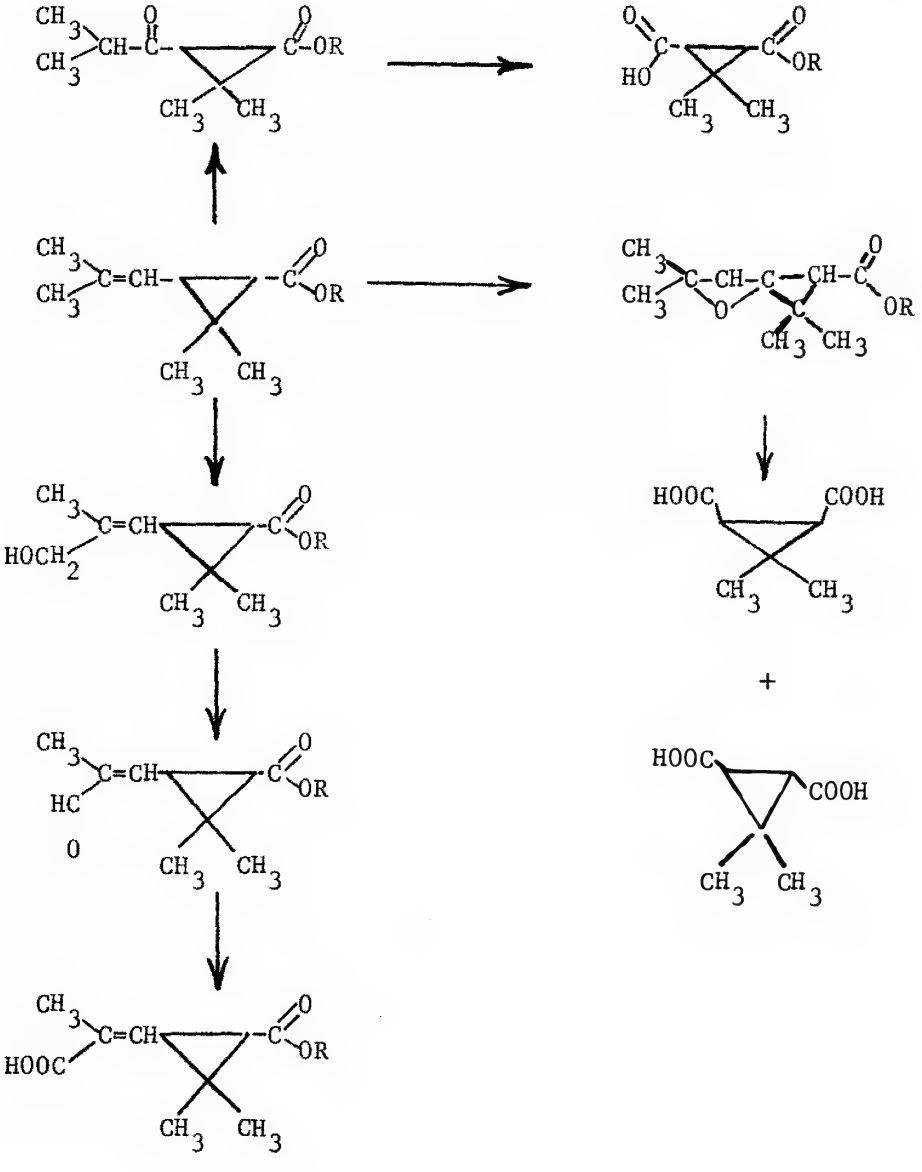
2,4-Dimethyl Hippuric Acid



2,4-Dimethyl Benzoyl Glucuronide

	$t_{1/2}$ hours
Allethrin	2.0
Dimethrin	3.6
Phthalthrin	1.0
Pyrethrin	0.2

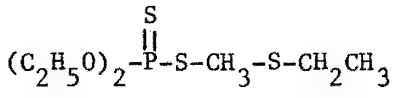
Photodecomposition of each of the four pyrethroids gave rise to twelve to sixteen products but little ester hydrolysis. Trans- and meso-cis-caronic acids were identified by co-chromatography (1638).



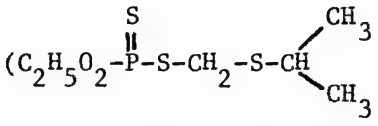
American Cyanamid 12008

(0,0-Diethyl S-isopropylthiomethylphosphorodithioate)

This compound is almost identical with thimet and appears to follow a metabolism route similar to thimet (150).



Thimet



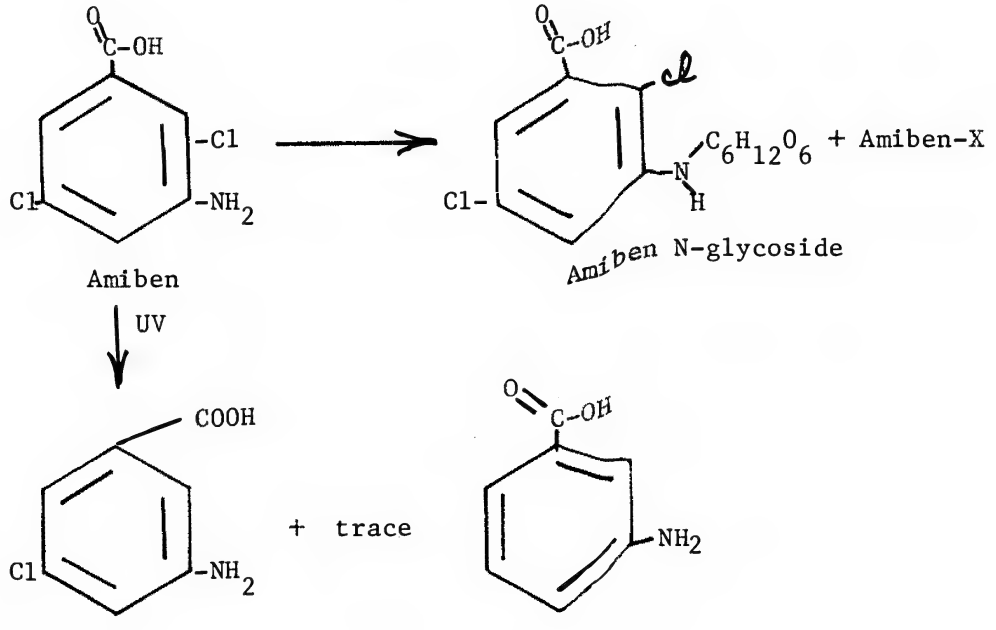
Am. Cyanamid 12008

Amiben (3-Amino-2,5-dichlorobenzoic acid)

The metabolism of amiben was studied in soybean plants (Glycine max (L.) Merr). Large amounts of the herbicide were taken up and disappeared rapidly. Of that which remained, most was present as a conjugate and could be released by alkaline hydrolysis. Infra-red, T.L.C., and G.L.C. were used to isolate and identify the complex formed. G.C. and I.R. analyses of the methylated hydrolysis products of the major metabolite demonstrated the presence of amiben. Similarly, the carbohydrate moiety was analyzed by G.C. and I.R. and shown to be D-glucose. The evidence that the carboxyl of amiben was not bonded to glucose in the metabolite and it was concluded that the metabolite was N-(3-carboxy-2,5-dichlorophenyl)-glucosylamine. It was also felt that a second compound obtained in these studies, first believed to be a minor metabolite, was an isomer or rearrangement product of N-glucosyl amiben and may be an artifact produced during extraction (294, 298, 1376, 1377, 1378). The N-glycoside was also isolated from barley (Hordeum vulgare L.).

Studies with carrots indicated that 4 or 5 compounds may be formed (39). In tomato and pigweed plants, conjugates of amiben were found. In the latter case, this has been identified as the N-glycoside (1377). Free amiben, as well as the N-glycoside, was found in susceptible broadleaf species and in all grass species studied except Johnson grass (1378, 1710).

Studies with soil microflora indicated that the breakdown of amiben occurred (1195) and that the carboxyl was cleaved slowly but steadily (917, 1499). Other studies showed that amiben underwent photo-chemical alteration when exposed to UV light (1170, 1285, 1784).



Amitrole (AT, ATA, 3-AT, Aminotriazole) (3-Amino-1,2,4-triazole)

Labeled amitrole (3-amino-1,2,4-triazole-5-C^{14}) was fed to rats. Analysis for radioactivity showed only traces of C^{14} in expired air. Seventy to ninety-five percent of the activity appeared in the urine within 24 hours as two unidentified metabolites and unchanged amitrole (430).

Tests indicated that 3-acetamido-1,2,4-triazole was not present (432). Feces contained small variable amounts of activity. After the first day, most internal organs exhibited some activity. No free amitrole was found in the liver. The average half-time for ATA clearance in various organs was 4.2 hours (3.4 to 5.1).

Studies have been conducted with amitrole applied to plants (251, 255, 652, 765, 1108), but metabolites were not identified. Other studies indicated that amitrole was activated to form a free radical, which was then capable of reacting with amino acids (233, 234, 235, 652, 654, 1005). Complexes of amitrole and alanine, glycine, or serine have been reported (230, 231, 232, 926, 927, 931, 933, 964, 1075, 1229, 1230). Infrared absorption spectral analyses and biochemical studies have also indicated that an amine glucoside was formed (411, 484, 485, 486, 487, 534) and that the amitrole-glucoside could be phosphorylated by yeast (488). In experiments with Abies concolor and Pinus ponderosa, amitrole was converted into three metabolic products, one of which seemed to be ATA-alanine (913).

Recent studies showed that the reproductive and vegetative structures of the bean plant, Phaseolus vulgaris, differed in their metabolism of amitrole. Vegetative parts formed stable complexes with the parent compound, whereas young reproductive organs cleaved the ring structure of amitrole and incorporated the carbon into common biochemical compounds such as, glucose, sucrose, fructose, and other compounds (1296). Bean plants transferred large quantities of C^{14} from serine C^{14} to 3-amino-1,2,4-triazolyl-1-alanine (3-ATAL) in both light and dark studies. Carbon from alanine C^{14}, and formate-C^{14}, also entered 3-ATAL but the percentage of conversion was far greater for serine. Yeast (Saccharmoyces cerevisiae) did not form 3-ATAL from 3-AT in 72 hours of incubation.

Toxicity of amitrole has been correlated with chemical analyses and it was found that biological response is proportional to the amount recoverable from soils (484, 1372). Studies have shown that amitrole not only partakes in the soil's base exchange system but that it also tends to form complexes with metals (249, 353, 354, 411, 1075).

The major metabolic product formed from amitrole by microbiological activity was carbon dioxide. At least thirteen additional unidentified compounds were also formed (38, 1210). E. coli converted 3-ATA into a metabolite, 3-amino-1,2,4,-triazolyl alanine. This was incorporated into cellular protein (1501). Studies with yeast showed that imidazolegylcerol phosphate accumulated, presumably because of histidine biosynthesis inhibition (662, 804, 1492).

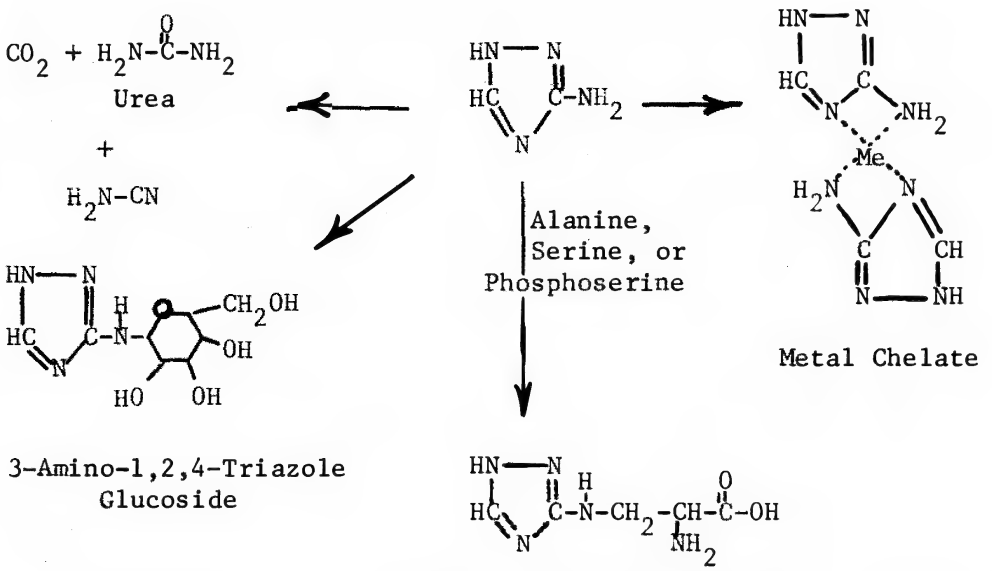
Plants were exposed to labeled ATA. The plant metabolites were then isolated and fed to rats. Plant metabolite-1 gave rise to two new spots and the absence of ATA. Thus, although metabolite-1 is further metabolized, it was not converted to ATA. Examination of radioautograms prepared from rat urine after feeding plant metabolite-2 did not show any new metabolites. The presence of ATA in the sample of metabolite-2 prevented drawing of conclusions as to whether or not this compound gave rise to ATA (432).

When exposed to UV (2200 A) amitrole broke down via free radicals and gave rise to CO_2, urea, and cyanamide (1171).

Conversion of Amitrole was first order and could be represented by

$$C_1 = C_o e^{-kt}$$

where k = 0.0371 for sugar beet leaves
0.0247 corn leaves
0.0299 bean leaves (426).



Antimycin

Female rats from the Holtzman strain were fed diets containing tritiated-antimycin. It was estimated that less than 15% of the total radioactivity in the urine was due to intact antimycin. Fractionation studies indicated that urinary metabolites could be subdivided: (1) Non-volatile 80%, volatile 20% --probably H^3-water; (2) Neutral, 15% of non-volatile--may or may not include intact antimycin; (3) Acidic plus neutral, 89% of non-volatile--products derived from aromatic portion of molecule but not containing basic nitrogen; up to 15% of this fraction possibly consisting of blastmycic acid; (4) highly water-soluble material, 11% of non-volatile (1213).

Arsenicals

Monosodium Methanearsonic Acid (MSMA)

Soil microorganisms degraded MSMA to CO_2 and arsenate. No other products were observed (1455).

After treatment of Johnsongrass (Sorghum halepense) with C^{14}-methanearsonic acid, two metabolites were observed. One appeared as an unidentified form of arsenic; the other was an unidentified complex (1279).

Azinphosmethyl (Guthion) [0,0-Dimethyl S-[4-oxo-1,2,3-benzotriazin-3-(4H)-ylmethyl] phosphorodithioate]

Using whole cockroach tissues and homogenates of these tissues, enzymes have been shown to be present that will activate guthion and degrade the active metabolite. By analogy to other studies, it is assumed that guthion is activated by oxidation to the corresponding phosphates. However, no metabolites have been identified (1060, 1067).

Using rat tissues, incubation of guthion with slices of brain, cardiac muscle, kidney, or skeletal muscal produced no active metabolite. When guthion was incubated with liver slices, an active anticholinesterase thought to be the oxygen analog was rapidly produced (385).

In the tissues and milk of cows fed guthion, four phosphorus-free metabolites, containing the benzazimide moiety, were found but not identified (421).

Activation and degradation of azinphosmethyl by liver microsomes of chicken, muscovey ducks, albino rats, mice guinea pigs, sheep, cattle and hogs was studied. Percent activation of azinphosmethyl (See table I) was calculated from the following formula:

$$pI_{50} = \frac{I_{50} \text{ directly assay P(O) analogue}}{I_{50} \text{ incubated insecticide}} X\ 100$$

$1 - pI_{50}$ = % degradation of P(O) azinphosmethyl.

pI_{50}

	Azinophosmethyl Activation		P(O) Azinophosmethyl Degradation	
	Male	Female	Male	Female
Mouse	7.59	7.55	8.02	7.83
Rat	7.73	7.47	8.23	8.28
Guinea Pig	7.65	8.09	7.59	8.00
Cattle	8.93	8.47	8.82	8.64
Hog	7.31	7.22	8.94	8.49
Sheep	8.89	9.00	9.01	9.07
Chicken	8.58	8.87	9.12	8.51
Duck	8.61	8.39	9.04	8.58

(737)

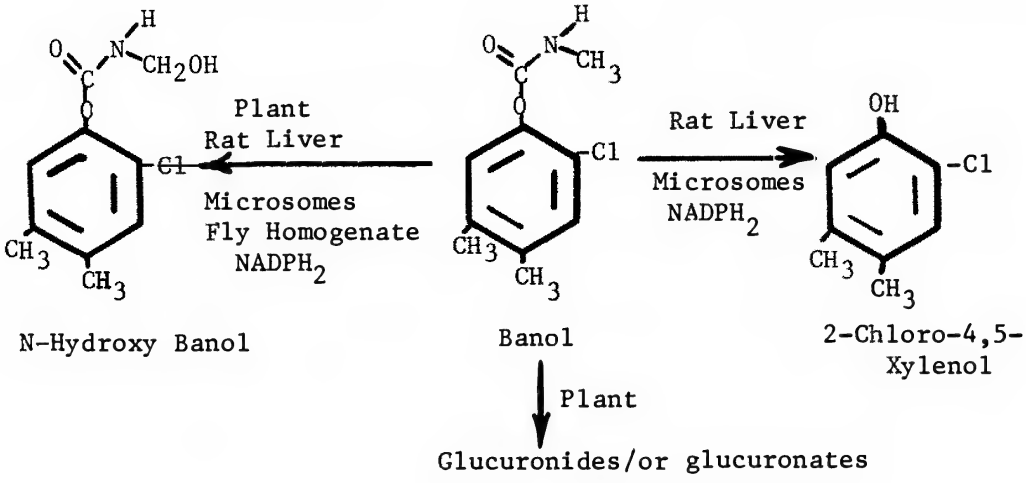
Banol (2-Chloro-4,5-dimethylphenyl N-methylcarbamate)

Following subacute oral administration of banol to rats, elimination was essentially complete within 48 hours. Methylamine to CO_2 were identified as metabolites. A number of other metabolites, including glucuronates, were observed but not identified (87, 836). In more recent studies 2-chloro-4,5-xylyl N-hydroxymethylcarbamate and 2-chloro-4,5-xylenol were identified after incubation of banol in a rat liver microsome-$NADPH_2$ system (1112).

Twelve day old pinto bean seedlings were placed in beakers containing labeled banol. Seven days later, the plants were macerated and extracted. Chromatography of the extracts revealed four metabolites. Although none of the metabolites were identified, one was characterized as a glucoside (496, 497, 845). In other studies N-hydroxy banol was identified. Seven other compounds were observed but not identified (846).

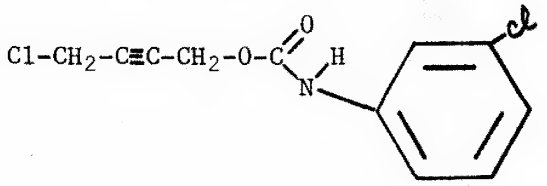
The degradation of banol by microsomes or by whole Blaberus giganteus fat body resulted in the formation of at least three metabolites. Studies indicated that one of these metabolites was N-hydroxymethyl banol (533, 1603). In houseflies, labeled $N\text{-}C^{14}H_3$ and $C^{14}{=}O$ gave rise to $C^{14}O_2$. Hydrolysis to the corresponding phenol also occurred (1001). In the presence of $NADPH_2$, fly homogenates also converted banol to the N-hydroxymethyl analog (1427).

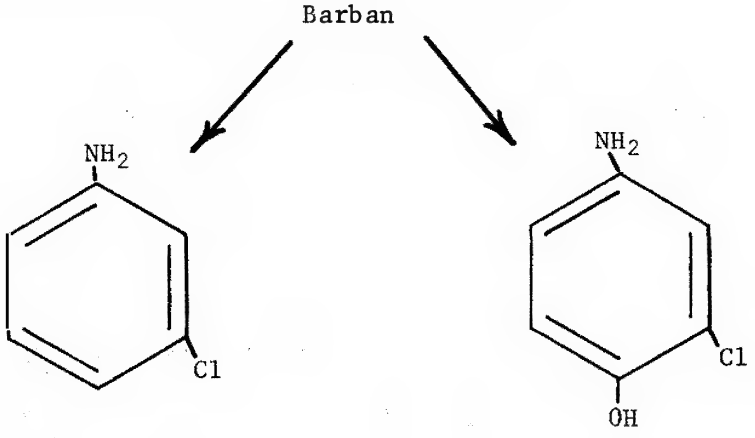
After exposure to UV (2537 A), 6 compounds were detected. Several unidentified compounds were also observed after treatment of bean plants (3).



Barban (4-Chloro-2-butynyl N-(3-chlorophenyl)carbamate)

After treatment with barban, plants formed a water-soluble 3-chloroaniline compound. Related carbamates were found to form similar products. Another metabolite detected, also in a bound state, was 2-chloro-4-amino-phenol (1209).





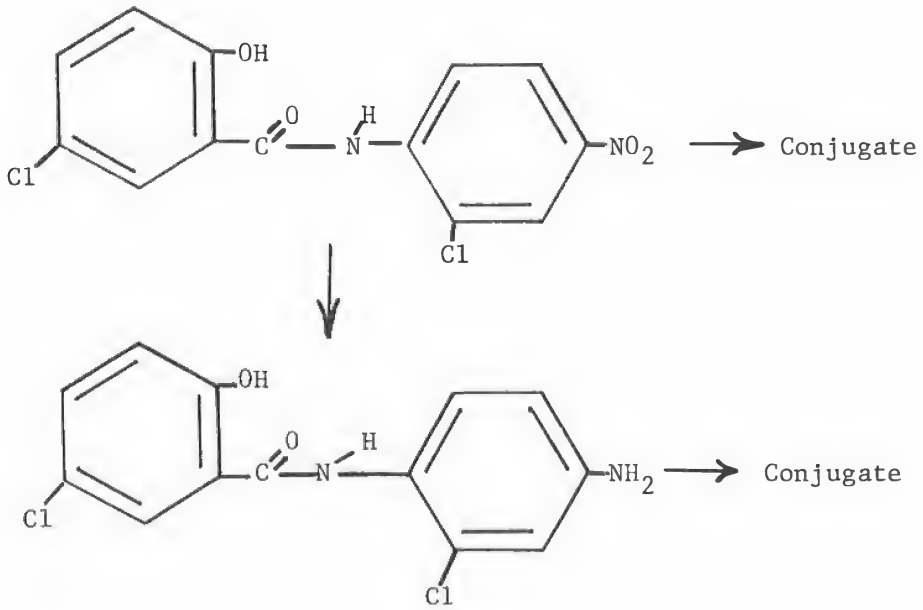
3-Chloroaniline

2-Chloro-4-Aminophenol

Bayer 73 (Ethanolamine salt of N-(2-chloro-4-nitrophenyl)-5-chloro-salicylamide)

Following oral administration of labeled Bayer 73 to rats, there was still 55% of activity present after one hour and 10% after five hours. About 33% of the applied activity appeared in the urine and 67% in the feces. Within 24 hours after administration, storage in tissues of rats was less than 1 ppm. Only 12% was detected in the urine of humans that ingested the material.

Unchanged active ingredient was excreted in the urine of rats in very small amounts as a conjugate. The main metabolite, N-(2-chloro-4-aminobenzene)-5-chloro-salicylamide, was also excreted in the urine in a conjugated form (388).



Bayer 9015 [3,3'-Dichloro-5,5'-Dinitro-o,o'-Biphenol]

After Bayer 9015 was fed to a lactating cow, only the intact compound was detected in feces. Residues were not detected in milk or urine. In the presence of rumen fluid, the compound rapidly disappeared. However, no metabolites were identified (1742).

Bayer 22408 (0,0-Diethyl 0-naphthalimido phosphorothioate)

P^{32} -labeled Bayer 22408 was applied dermally as a 0.5% emulsion to Holstein cows. The intact insecticide was found in the milk for 6 days. The oxygen analog was not found in milk but constituted the predominant non-hydrolyzed product in the feces (213).

Baygon (Bayer 39007) (o-Isopropoxyphenyl N-methylcarbamate)

Incubation of baygon with rat liver microsomes gave rise to about seven metabolites. Those identified were: 2-isopropoxyphenyl N-hydroxymethylcarbamate; 2-hydroxyphenylmethylcarbamate; 2-isopropoxyphenol; 2-isopropoxyphenyl carbamate; and 2-isopropoxy-5-hydroxyphenylmethylcarbamate. Carbon dioxide was also evolved, probably from the methyl group (352, 836, 1110, 1112).

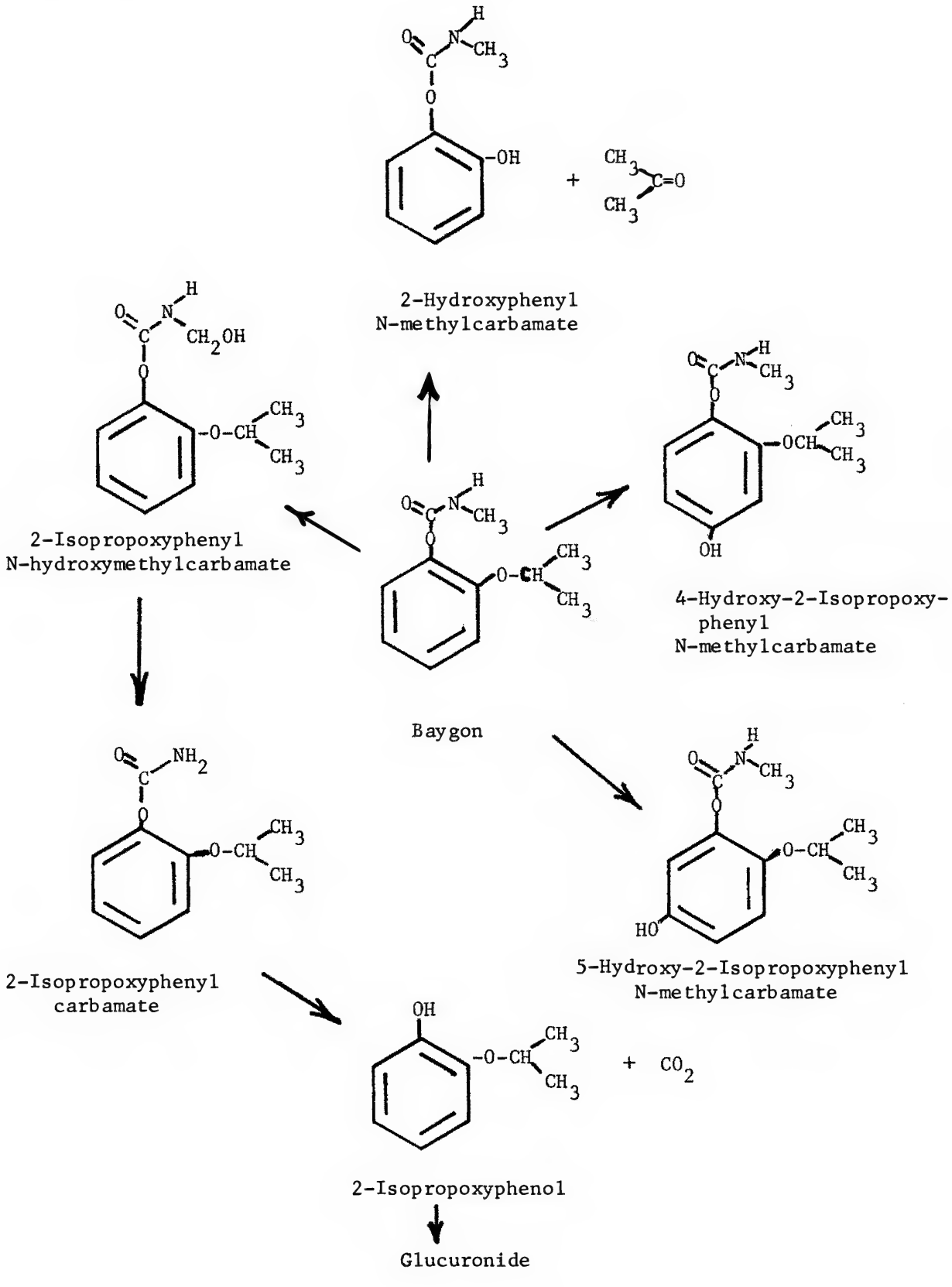
When fed to humans, baygon was recovered from urine almost quantitatively as o-isopropoxyphenol, probably excreted as the glucuronide (352).

The N-hydroxy analog, o-isopropoxyphenol and 2-hydroxyphenylmethylcarbamate were observed after exposure of houseflies and roaches to baygon (352, 1001, 1111, 1427). In houseflies, labeled N-$C^{14}H_3$ and C^{14}=O gave rise to $C^{14}O_2$. Carbon dioxide was also formed rapidly by metabolism of the C^{14}-labeled isopropoxy group (1001). In other studies with susceptible and baygon resistant strains of Musca domestica L., hydrolysis was shown to be unimportant as a metabolic reaction. The attack on baygon involved hydroxylation; and the metabolites, which were the primary hydroxylation products or the result of the degradation of these materials, were identified as (in order of their decreasing amounts): 5-hydroxy-2-isopropoxyphenyl methylcarbamate, 2-hydroxyphenyl methylcarbamate and acetone, 2-isopropoxyphenyl N-hydroxymethylcarbamate, and 2-isopropoxyphenyl carbamate. There were six or more additional unidentified compounds. The metabolites with hydroxyl groups were conjugated as evidenced by the release of the hydroxylated carbamates when feces extracts were incubated with β-glucosidase, β-glucuronidase, sulfatase, and acid phosphatase. The 5-hydroxy baygon predominated (1703).

Plants converted baygon into a water soluble fraction (378). In bean plants, in addition to seven unidentified metabolites, 2-hydroxyphenylmethylcarbamate, 4-hydroxy baygon, and N-hydroxymethyl baygon were identified (845, 846).

Twenty-five percent of the applied baygon was lost from sand in 100 days, but there was practically no decline in silt-loam soil during a six month period (579).

When exposed to ultra-violet light (2537 A), baygon decomposed but the products were not identified (3).


2-Hydroxyphenyl
N-methylcarbamate
+
2-Isopropoxyphenyl
N-hydroxymethylcarbamate
4-Hydroxy-2-Isopropoxy-
phenyl
N-methylcarbamate
Baygon
2-Isopropoxyphenyl
carbamate
5-Hydroxy-2-Isopropoxyphenyl
N-methylcarbamate
+ CO2
2-Isopropoxyphenol
Glucuronide

BHC (Benzene hexachloride) (1,2,3,4,5,6-Hexachlorocyclohexane)
(Lindane = γ-BHC)

Radiochemical methods used to study the fate of γ-BHC in flies showed that no respired gases contained C^{14}. Although most of the radioactivity could be extracted by carbon tetrachloride, an unextracted residual activity remained in both resistant and normal flies. In addition to pentachlorocyclohexene, some water-soluble metabolites were found (157, 158, 159, 160, 162, 179, 712, 1114, 1357). Other studies indicated, however, that the first step in the metabolism of γ-BHC was the removal of one chlorine atom and the formation of a C-S bond, followed by loss of additional chlorine atoms and the formation of all six isomers of dichlorothiophenol (163). Anopholes gambiae were also able to convert α- and γ-BHC to water-soluble compounds (161). Recently, two isomers of pentachlorocyclohexene were obtained after exposure of houseflies to γ-BHC (1587).

When γ-BHC was fed to dairy cows (1465), no pentachlorocyclohexene was found in the milk. Studies with dogs, rabbits, and rats indicated a rapid breakdown of BHC into 1,2,4-trichlorobenzene and other compounds of unknown structure (40, 183, 304, 825, 1264). When labeled α- and γ-BHC was administered to rats intraperitoneally, a high concentration of radiating material was present in the central nervous system. Both isomers were dechlorinated *in vivo* and converted to water soluble compounds which were excreted by the kidneys (824). In rats, urinary excretion reached a peak on the 5th to 8th day after administration and BHC was not detected after 17-18 days (1252, 1551). A single oral administration of 50 or 100 mg of γ-BHC/kg body weight of rats increased levels of total glucuronic acid excreted in urine during 14-15 days by a daily average of 1.5 mg, and organic sulfur compounds by 35-58% during 13-14 days. Thin layer and silicic acid column chromatography showed undecomposed γ-BHC and 2 metabolites in urine and liver for 14 days after administration of the BHC (1251).

γ- and δ-1,3,4,5,6-pentachlorocyclohexene-1 were metabolized by rat and liver microsomes in the presence of $NADPH_2$ and O_2 (711). In other studies, γ-BHC and γ-2,3,4,5,6-pentachlorocyclohex-1-ene were converted by rats into 2,3,5,- and 2,4,5-trichlorophenol and excreted in urine as free phenols, sulfates, and glucuronic acid conjugates. 2,4-Dichloromercapturic acid was also isolated (570). The products found in these studies resemble those in the metabolism of 1,2,4-trichlorobenzene in rabbits (742), and it would appear that the metabolism of γ-BHC proceeds via γ-2,3,4,5,6-pentachlorocyclohex-1-ene to 1,2,4-trichlorobenzene. In other studies, α-BHC gave rise to 2,4,6-trichlorophenol (823).

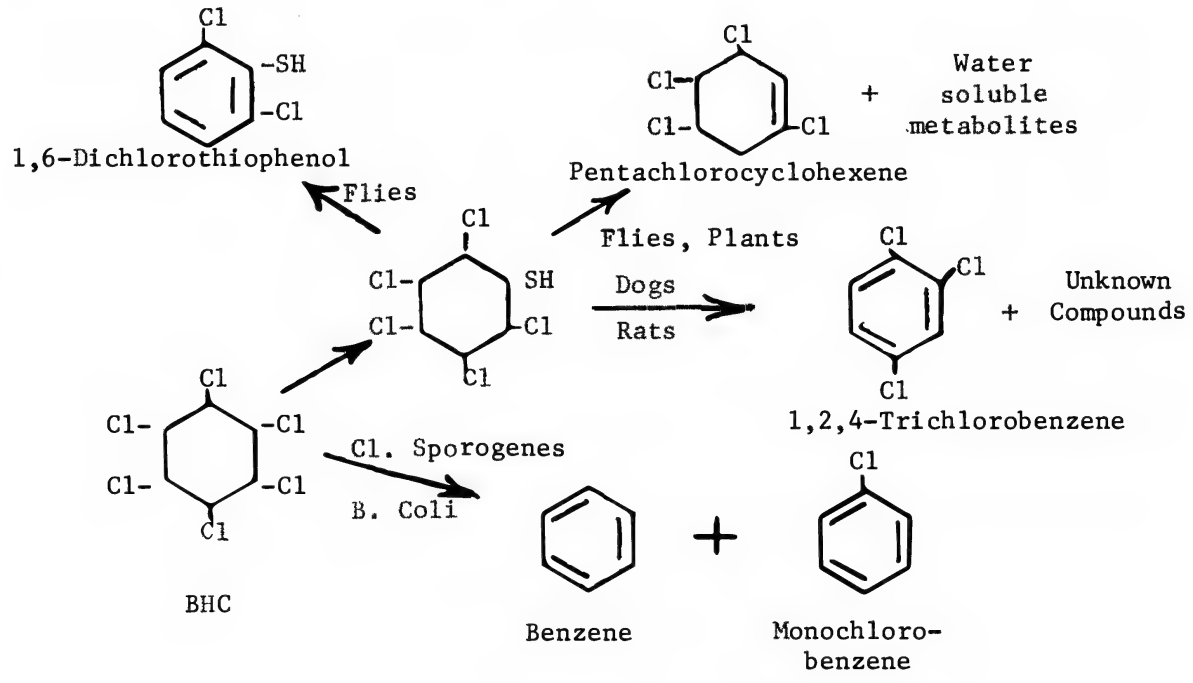
Experiments with plants were inconclusive but the data indicated pentachlorocyclohexene as a metabolite (132, 1264). In soil, lindane broke down rapidly to a non-toxic compound (164, 869) which was identified as γ-pentachlorocyclohexene (1534). Some CO_2 was also formed from γ-BHC in submerged soils (919).

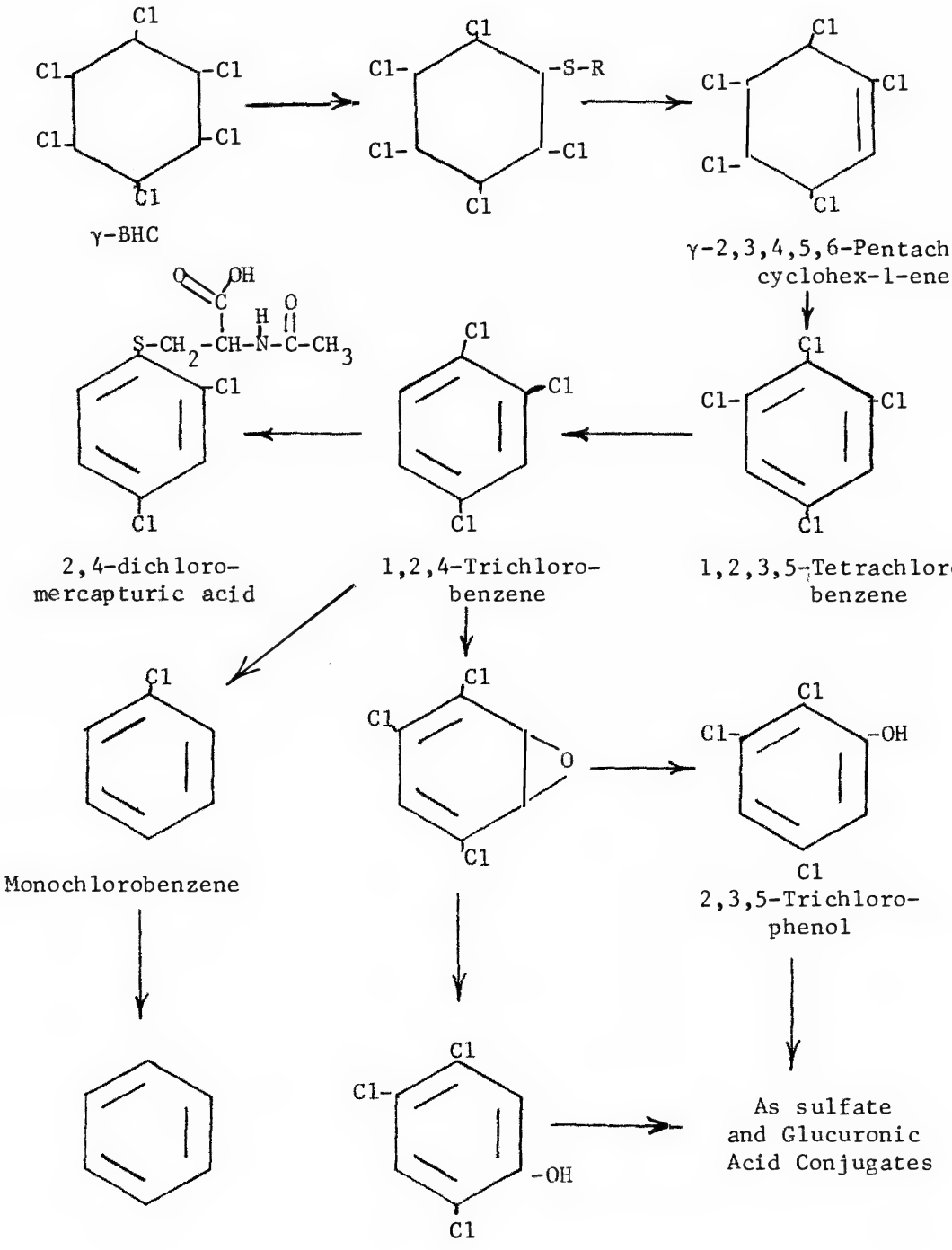
When exposed to ultra-violet irradiation (2537 A), α-,β-,γ-, and δ-BHC did not yield any products (1228).

When cattle dips were imitated in the laboratory by combining soil, feces, urine and extracts of scrapings from cattle hides, the γ-isomer was lost much more rapidly than other isomers. Using the bacteria Clostridium sporogenes or Bacillus coli found in such mixtures, BHC gave rise to traces of benzene in seven days as well as monochlorobenzene (17). The cattle tick, Boophilus decoloratus, gave rise to S-(2,4-dichlorophenyl) glutathione (280).

C^{14} labeled α-BHC was used to study the effect of heating at baking temperatures in flour. About 90% of residual activity was 1,2,4-trichlorobenzene. Small amounts of 1,2,3- and 1,3,5-trichlorobenzene, the three dichlorobenzenes, chlorobenzene and possible benzene were also present (178).

In sea water, the gamma isomer apparently "decayed" more rapidly than the alpha or beta isomers. No breakdown products were identified (1601).




γ-BHC
γ-2,3,4,5,6-Pentachloro-cyclohex-1-ene
1,2,3,5-Tetrachloro-benzene
1,2,4-Trichloro-benzene
2,4-dichloro-mercapturic acid
Monochlorobenzene
2,3,5-Trichloro-phenol
As sulfate and Glucuronic Acid Conjugates

Bidrin (3-Hydroxy-N,N-dimethylcrotonamide dimethylphosphate)

Azodrin (3-Hydroxy-N-methylcrotonamide dimethylphosphate) (N-methyl bidrin, SD 9129).

Using P^{32} -labeled bidrin and rats, 83% of the recovered activity after injection was in the form of dimethylphosphate after 20 hours. Metabolites were excreted rapidly. Within 24 hours, 83% of the injected dose had been excreted. Extensive oxidation occurred with the formation of hydroxymethyl bidrin and N-methyl bidrin (azodrin). Subsequent hydrolysis gave rise to dimethyl phosphate, bidrin acid, desmethyl bidrin acid, monomethylphosphate, and finally inorganic phosphate. Hydrolysis of bidrin itself gave rise to desmethyl bidrin (199).

When administered orally to rats, 63-71% of the P^{32} was excreted in 48 hours. No marked sex or compound differences were noted. Most of the radioactivity was excreted in urine within 6 hours. An additional 5-6% appeared in the feces within 48 hours following treatment. Residual P^{32} in the tissues did not vary with sex. An additional 12% was recovered as $C^{14}O_2$ within 48 hours after treatment of male rats with bidrin-N-methyl-C^{14} (984, 983).

Urinalyses on dogs, mice, and rabbits, as well as on rats and goats, indicated that species variation existed in the rate at which bidrin-P32 was metabolized and the extent of metabolite excretion in the urine (984).

Significant amounts of N-hydroxymethylamide analog of azodrin appeared following administration to rats of either bidrin or azodrin while the N-methyl-N-hydroxymethylamide compound was present only following bidrin treatment, and the unsubstituted amide only following azodrin treatment (984).

In rats administered azodrin only trace amounts of azodrin acid were detected in extracts, indicating minimal hydrolytic degradation by amidase action. Dimethyl phosphate and O-demethyl azodrin were the major degradation products of azodrin. It was felt that degradation followed formation of hydroxymethyl azodrin rather than loss of HCHO. The finding of only trace amounts of N-demethyl azodrin and the stability of the pure compound seemed to indicate that demethylation was a minor reaction (200).

Goats treated orally with bidrin or azodrin excreted in the urine a high proportion of the administered P^{32} or N-methyl-C^{14} labels. Trace amounts of bidrin, azodrin, the N-hydroxymethylamide analog of azodrin, and the unsubstituted amide were present in the urine (984). Labeled compounds appeared in the goat milk after treatment with either P^{32} or

C-^{14}-labeled bidrin or azodrin. No N,N-dimethylacetoacetamide, N-methylacetoacetamide, or 3-hydroxy-N,N-dimethylbutyramide was present in the milk and the nature of the C^{14} materials remained unknown. The major organoextractable P^{32} material in the milk following both bidrin and azodrin treatment was azodrin. Traces of the N-methyl-N-hydroxymethylamide and the unsubstituted amide were also present (984).

Adult boll weevils and fifth-instar bollworm larvae were treated with labeled bidrin. Metabolites recovered were similar to those recovered from rats (199). Houseflies and American cockroaches rapidly metabolized bidrin and azodrin to form the N-hydroxymethyl and N-demethylated derivatives. Comparatively large amounts of the N-hydroxymethylamide compounds were detected in flies treated with bidrin or azodrin alone but they were absent from extracts of flies treated with the toxicant plus sesamex; and only minute amounts of azodrin were detected in flies treated with bidrin plus sesamex (601, 984).

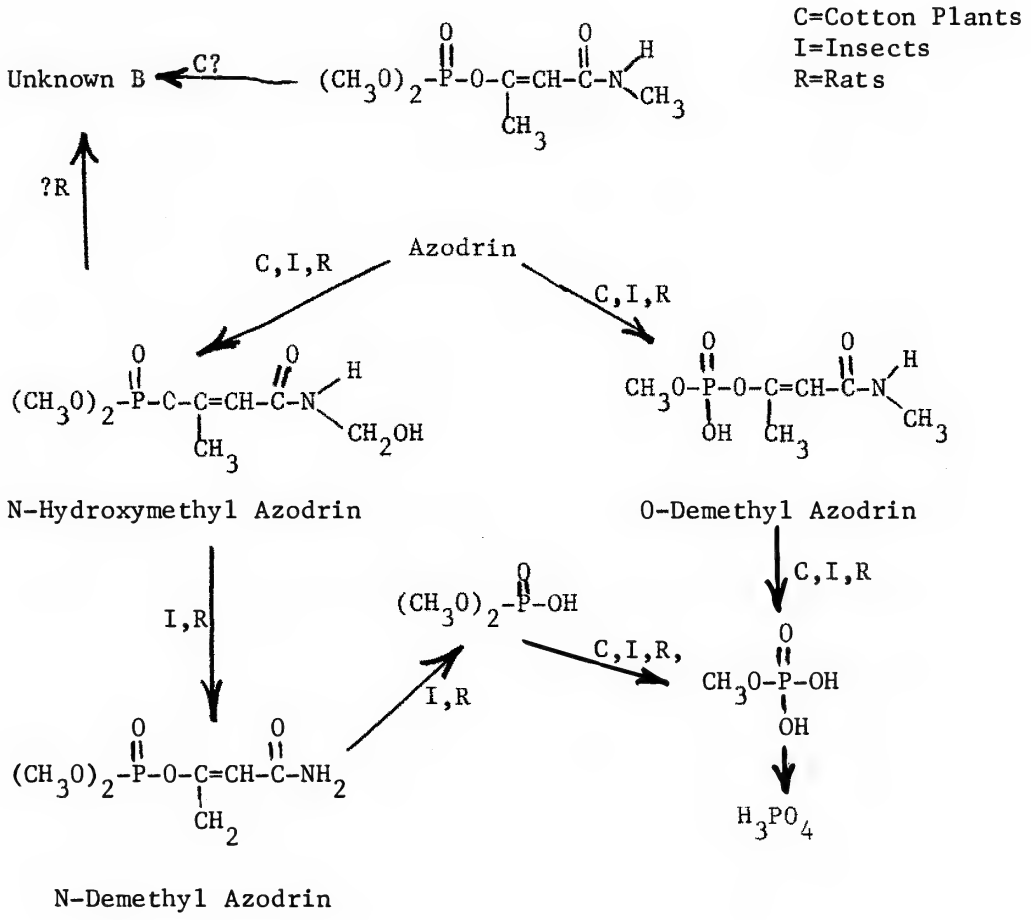
In other studies absorbed azodrin was metabolized more rapidly in houseflies than in weevils. Hydroxymethyl azodrin was detected after 4-8 hours in houseflies but not until after 24 hours in weevils. Dimethyl phosphate was the major hydrolysis product in houseflies; O-demethyl azodrin predominated in weevils (200).

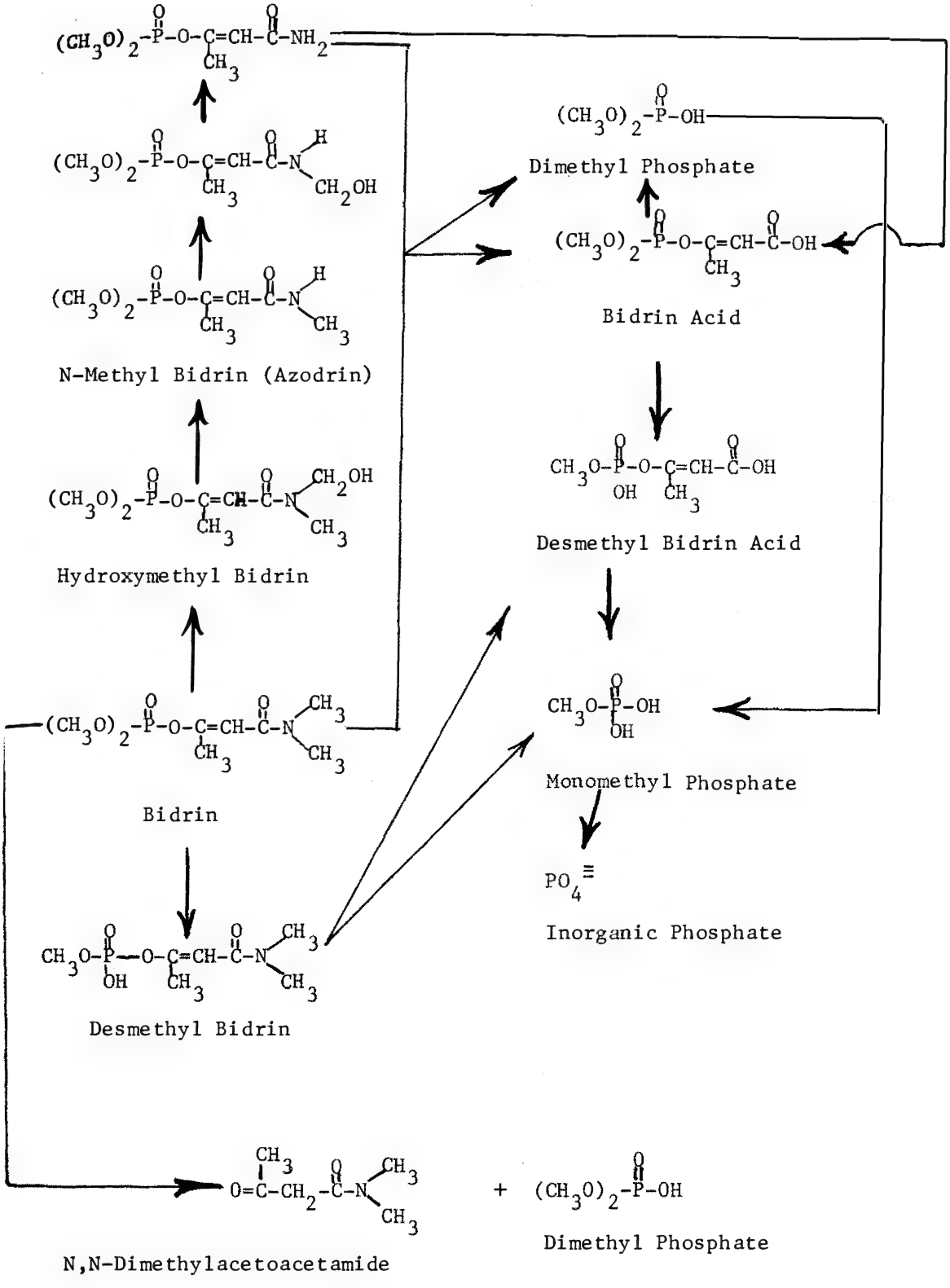
When cotton plants and leaves were treated with bidrin, initial detoxification included hydrolysis of the vinyl-phosphate bond to form dimethylphosphate and N,N-dimethylacetoacetamide; cleavage of a methylphosphate bond to form desmethyl bidrin and methanol; and hydrolysis of the amide bond to form bidrin acid and dimethylamine. One plant metabolite was not identified, leaving a gap in the metabolic path of bidrin in plants (199).

After injection into the stems, bidrin-P^{32} and azodrin P^{32} were rapidly translocated in bean plants and persisted for several weeks under greenhouse conditions. Half-life values for bidrin and azodrin were 9 and 14 days respectively. Administration of bidrin yielded azodrin and traces of the N-methyl-N-hydroxymethylamide. Azodrin was identified by I.R. Following azodrin administration, traces of the mono-N-hydroxymethylamide and the unsubstituted amide were present at eight days but only azodrin was present after twenty days (984).

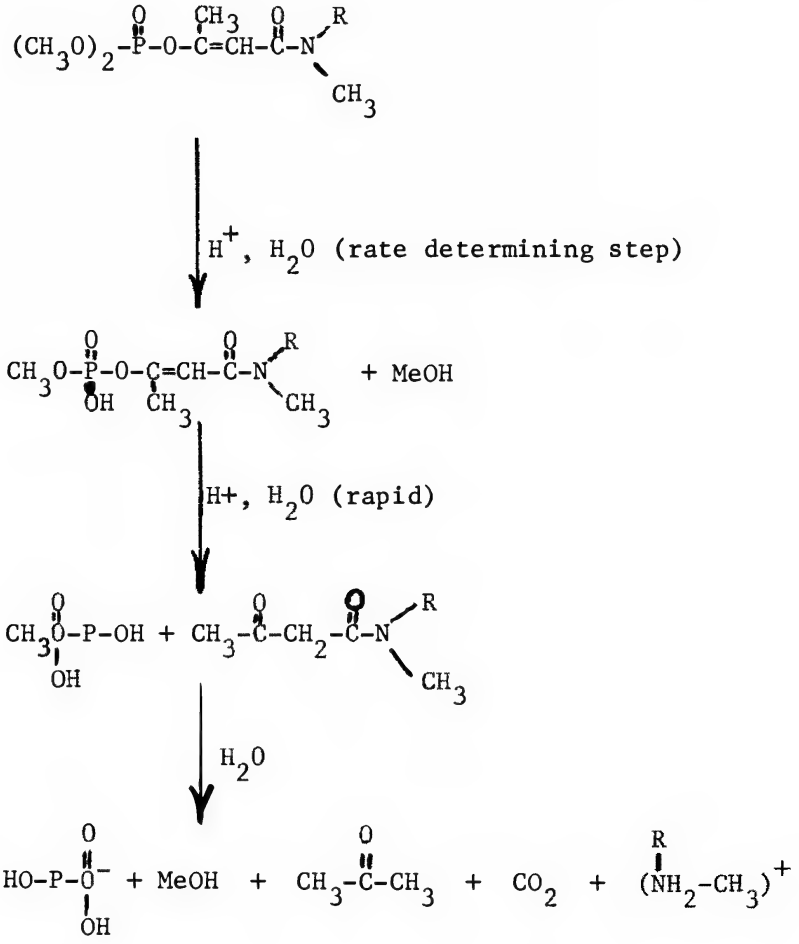
The same pathway occurs, at least in part, in all organisms tested. The relative amounts of the metabolites were dependent on the experimental organism and the administered compound. In hen eggs injected with bidrin, all products except the N-methyl-N-hydroxy methylamide of bidrin were detected (984).

In moist soil, bidrin leaches readily and breaks down rapidly (307). One week after planting of seeds treated with P^{32}-azodrin, 50% of the azodrin was still present. Over a period of 21 days, orthophosphate, dimethyl phosphate, O-demethyl azodrin, and N-hydroxymethyl azodrin were detected. An unidentified compound was observed and some of the label was unextractable. The loss of azodrin from cotton leaves, following foliar application, was for the most part probably the result of volatilization. The same metabolites were detected as from the seed treatment experiments except that an additional unidentified compound was observed (876).



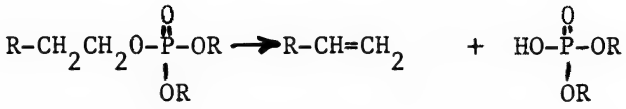

Dimethyl Phosphate
Bidrin Acid
N-Methyl Bidrin (Azodrin)
Desmethyl Bidrin Acid
Hydroxymethyl Bidrin
Monomethyl Phosphate
Bidrin
Inorganic Phosphate
Desmethyl Bidrin
N,N-Dimethylacetoacetamide
Dimethyl Phosphate

Under acid conditions of hydrolysis, methanol, acetone, orthophosphoric acid, methyl phosphoric acid, and N,N-$(CH_3)_2$-acetoacetamide were all identified. Some dimethylamine was probably also present. Thus, acid hydrolysis proceeded by initial loss of a methoxy-group followed by rapid loss of the crotonamide. Some chromatograms suggested the formation of desmethoxy bidrin or azodrin, both high unstable in acid solution. Even after 6 hours of refluxing, significant quantities of dimethylacetoacetamide were present. The stability of the amide group in bidrin indicated that 3-(dimethoxyphosphinyloxy)crotonic acid and its des-methoxy derivatives were not part of the major decomposition path (193).

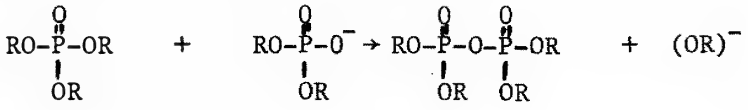


R= Methyl (Bidrin)
or= H (azodrin)

Alkyl and aryl phosphates decomposed slowly under the influence of heat. The reaction was catalyzed by UV and acid (193).

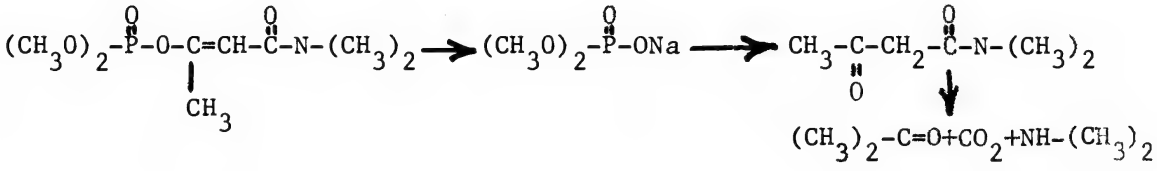


Orthophosphate esters underwent a different reaction (520).



During alkaline hydrolysis of bidrin, the crotonamide moiety separated from the phosphate. This was followed by decomposition of the crotonamide to acetone, CO_2 and di-or-mono-methylamine.

The dimethyl phosphate ion was stable under alkaline conditions and was not further hydrolyzed.



Thermal decomposition

$$K = Ae^{-\frac{E}{RT}}$$

K= rate of reaction

A= constant

E= activation energy

R= gas constant

T = ^{o}K

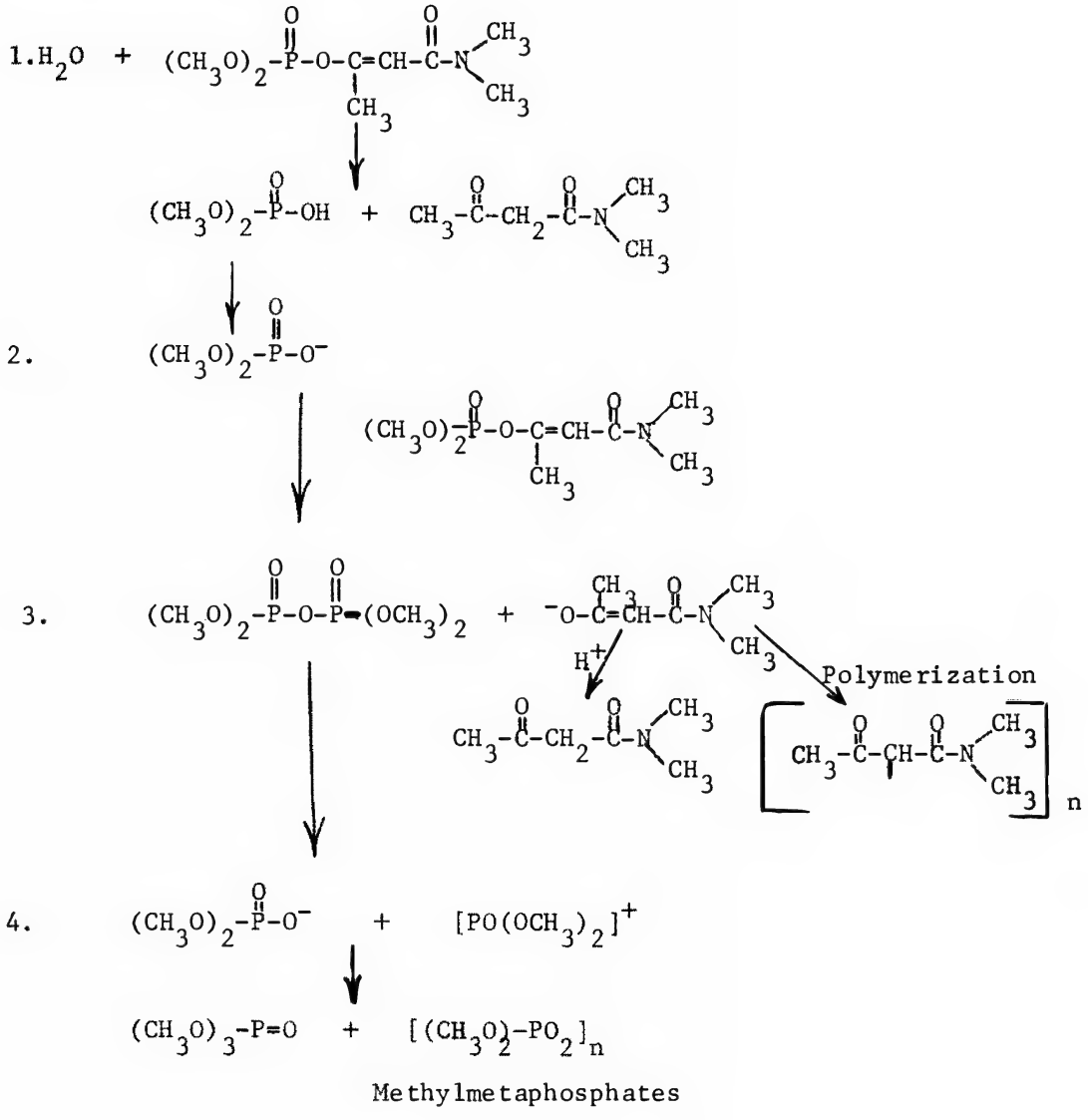
$$\ln K = \ln A - \frac{E}{RT}$$

Calculation showed that at 55°C the rate of decomposition of bidrin became appreciable. Infra-red analyses showed the presence of trimethylphosphate, N,N-dimethylacetoacetamide, dimethylamine salt of H_3PO_4, polyphosphate, and a compound formed by condensation of two or more crotonamide residues. Thus, thermal decomposition of bidrin initially produced orthophosphoric acid and its methyl esters. A pyrophosphate esterified with methyl groups also seemed to be present.

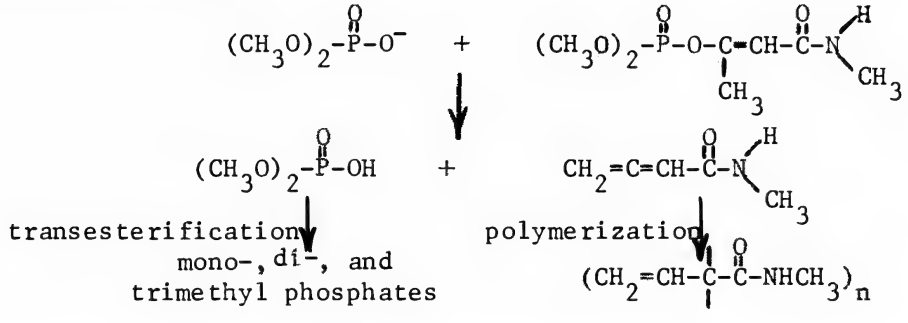
Technical bidrin contains measurable amounts of strong acid, mainly dimethyl phosphoric acid, and water. On storage, the water gradually hydrolyzes the di-ester to produce further acids. When the concentration of acid reaches a sufficient level, a self sustaining trans-esterification reaction between phosphoric acids and insecticide can be initiated (193).

Studies with azodrin indicated stability of the crotonamide group and a mechanism for decomposition different than that for bidrin (193).

Bidrin Decomposition



Azodrin Decomposition

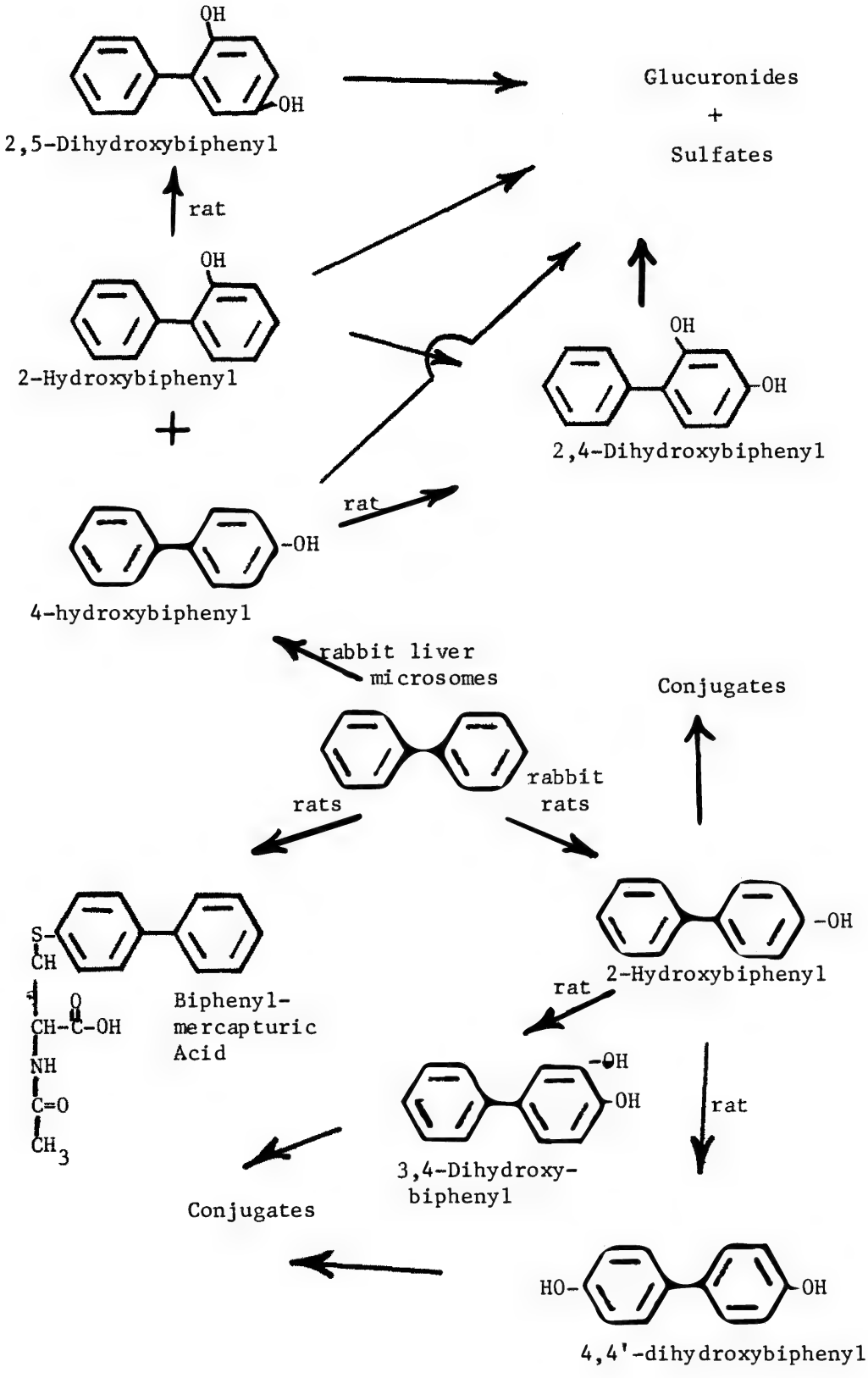


Biphenyl, 4-Chlorobiphenyl, and 2-Hydroxybiphenyl

After rabbits ingested biphenyl or 4-chlorobiphenyl, 64% and 65%, respectively, was accounted for in the urine primarily as the corresponding glucosiduronic acid and smaller amounts of phenols and ether sulfates. From the urine, 4-hydroxybiphenyl and 4-biphenyl glucosiduronic acid were isolated and identified after ingestion of biphenyl. After ingestion of 4-Chlorobiphenyl, 4-(p-Chlorophenyl) phenol and the corresponding glucosiduronic acid were also isolated from the urine. The ethereal sulfates were not identified (128). In the urine of the rat, 4-hydroxy-, 4,4'-dihydroxy- and 3,4-dihydroxybiphenyl and their conjugates and biphenylmercapturic acid were found (1490).

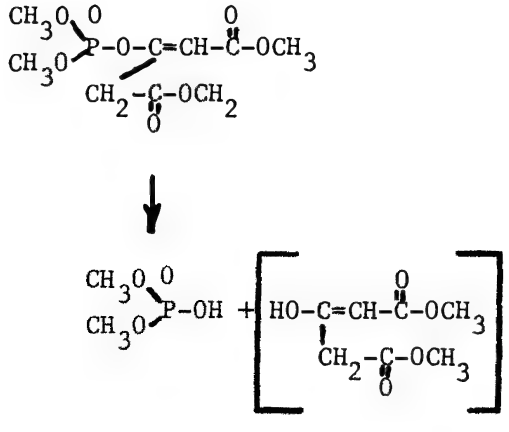
Liver microsomal preparations from New Zealand white rabbits converted biphenyl into 2- and 4-hydroxybiphenyl (1611). Other studies with rabbits, indicated that the 4-isomer and practically no 2-isomer were found (313). The extent of 4-hydroxylation varies with species being poor in cat and trout and high in mouse and coypu. Ability to form 2-hydroxybiphenyl is almost absent in livers of adult rabbits and rats, guinea pigs, hens, trout, and fox. Livers of mice, hamsters, cats, coypus, frogs, and young rabbits and rats form measurable amounts of the 2-isomer (313).

2-Hydroxybiphenyl, orally administered to rats, was excreted in the urine as the gluronides and sulfates of 2-hydroxybiphenyl and the metabolite 2,5-dihydroxybiphenyl. Traces of another metabolite, thought to be the polyhydroxy compound 2,4-dihydroxybiphenyl, were observed but identification could not be made (415).


OH
OH
2,5-Dihydroxybiphenyl
Glucuronides
+
Sulfates
rat
OH
2-Hydroxybiphenyl
+
OH
OH
2,4-Dihydroxybiphenyl
rat
-OH
4-hydroxybiphenyl
rabbit liver
microsomes
Conjugates
rabbit
rats
rats
S-
CH
CH-C-OH
NH
C=O
CH3
Biphenyl-
mercapturic
Acid
-OH
2-Hydroxybiphenyl
rat
-OH
OH
3,4-Dihydroxy-
biphenyl
rat
Conjugates
HO-
OH
4,4'-dihydroxybiphenyl

Bomyl (GC-3707) [Dimethyl 3-(dimethylphosphate)glutaconate]

In the presence of mouse liver enzymes and glutathione, cis- or trans-Bomyl was hydrolyzed with production of dimethyl phosphate (1037).



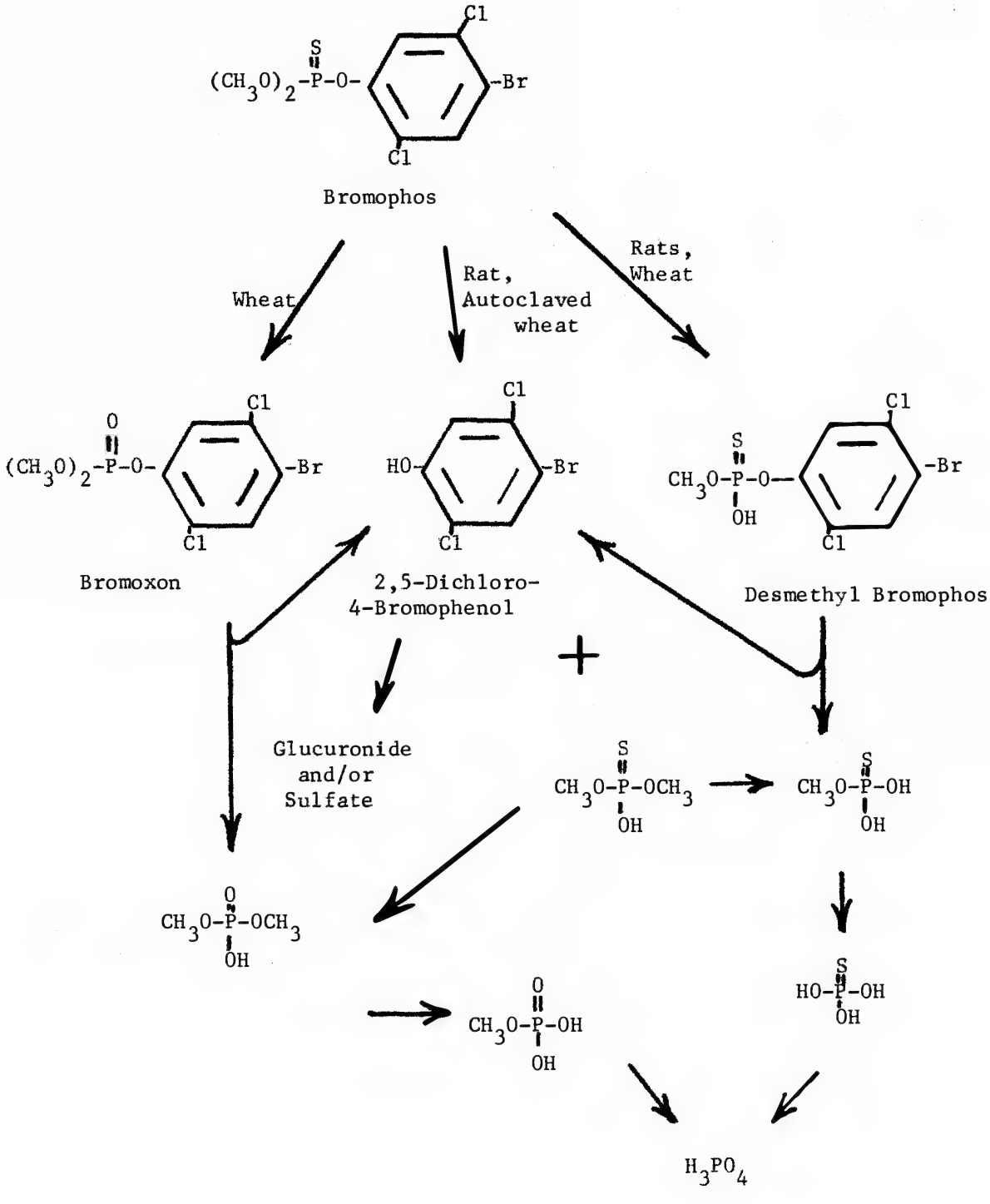
Bromophos (O-(4-Bromo-2,5-dichlorophenyl) O,O-dimethylphosphorothioate)

Labeled bromophos was administered orally to rats. Absorption was good and elimination practically complete after 24 hours. There was no accumulation in examined organs. Some inorganic phosphate was incorporated into the organism phosphate pool. The phenolic moiety of bromophos was excreted rapidly in urine and feces as the glucuronide or sulfate. Six metabolites were detected in the urine of rats: phosphate, dimethyl thionophosphate, monodesmethylbromophos, dichlorobromophenol, and two unidentified compounds. Neither bromophos, bromoxon, nor monodesmethylbromoxon were found in the urine (1362).

Bromophos was applied to stored wheat grains and the decomposition studied over a 10 week period. Although only traces of dimethyl phosphorothionate were found, indicating that desmethyl bromophos is the main phosphatase degradation product, large amounts of monomethylphosphorothionate were detected. Bromoxon was found only during the first 20 days in amounts up to 10% of the original bromophos level (1246).

Degradation of bromophos proceeded rapidly for about 7 days. Degradation then stopped for about 3 weeks because of accumulation of desmethyl bromophos, which inhibits phosphatase hydrolysis of bromophos. After the desmethyl bromophos had been degraded, bromophos degradation resumed. The level of free dichlorobromophenol did not increase significantly until degradation of desmethyl bromophos was well under way, indicating formation of the phenol from the desmethyl or oxon compounds. When bromophos was applied to autoclaved grains, only dimethyl phosphorothionate and dichlorophenol were produced (1246).

Oxidation of bromophos to bromoxon occurred in the seed coats and germs of wheat grains. Hydrolytic activity was found in the germ and endosperm (1247).


Bromophos
Wheat
Rat,
Autoclaved
wheat
Rats,
Wheat
Bromoxon
2,5-Dichloro-
4-Bromophenol
Desmethyl Bromophos
Glucuronide
and/or
Sulfate
H_3PO_4

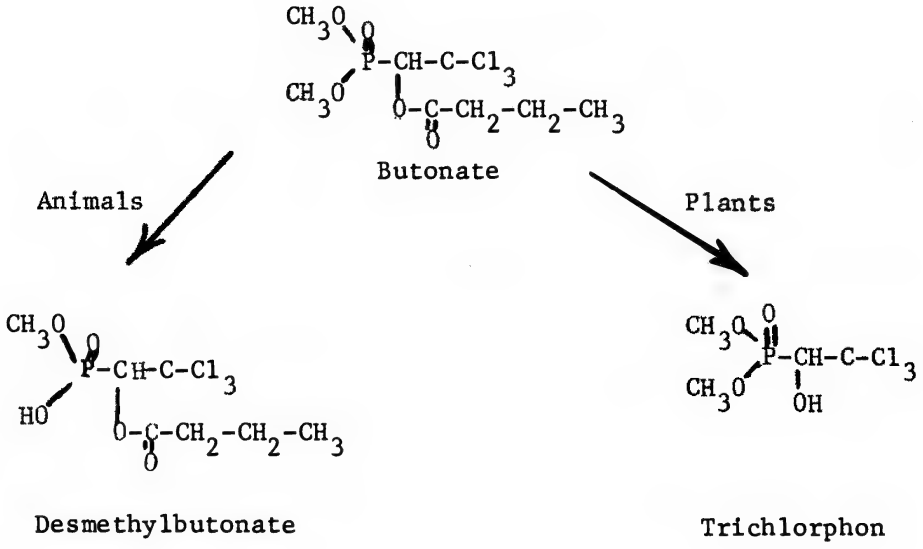
Butonate (Tribuphon) [0,0-Dimethyl-1-butryloxy-2,2,2-trichloroethylphosphate]

In the liver and blood of warm blooded animals, butonate was degraded primarily by hydrolysis of P-C bond with formation of dimethylphosphate. Small amounts of the desmethylbutonate also formed in the blood in vitro (1562, 1730). Butonate was also hydrolyzed by fish (1681).

In insects, the organic acid was removed by esterase action and trichlorofon (dipterex) was formed, which in turn was metabolized to DDVP (1562).

After exposure of plants to butonate, trichlorofon was observed. Twenty-four hours after application, the trichlorofon amounted to 55-65% of the applied dose on apples, 50%, on plums; 17-25%, on cherries; 5%, on peas; 65% on immature wheat; and 18%, on mature wheat. The half-life on apples and plums was 1.5 days; on cherries, 0.5 days (1562).

See also DDVP and Trichlorofon (Dipterex)



Captan (N-Trichloromethylthio-4-cyclohexene-1,2-dicarboximide)

The intercellular breakdown product of captan, thiophosgene, is capable of inhibiting certain enzyme systems that have cocarboxylase as a coenzyme (387, 692, 797, 911). Captan reacted with sulfhydryl compounds, in general, such as cysteine and glutathione (889, 1121, 1208, 1310). The reaction does not occur below pH 4.0 or 5.0, respectively, and the rate rises rapidly above pH 6.0 (889). Products of the reaction with cysteine were cystine, tetrahydrophthalimide, hydrogen sulfide, 2-thiazolidinethione-4-carboxylic acid, HCl, and what was then believed to be carbon disulfide. The trichloromethylthio group and thiophosgene were apparently intermediates in the formation of 2-thiazolidinethione-4-carboxylic acid. From aqueous solutions of thiophosgene, a compound with the melting point of di(thiophosgene) was isolated (912). Recent studies have indicated that the lack of specificity of Viles solution led to the erroneous identification of one metabolite as carbon disulfide. Infrared and gas chromatographic analyses have shown that this compound, derived either from the reaction of captan and glutathione or from the reaction of captan with N. crassa spores, was in fact carbonyl sulfide (1336).

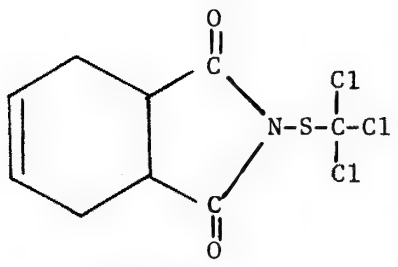
Although captan did not appear capable of reacting with other than sulfhydryl groups, the trichloromethyl group of phosgene released from captan by sulfhydryl groups was apparently capable of reacting with amino, hydroxyl, sulfhydryl, and possibly other groups. Histidine and serine reacted with thiophosgene to form UV-absorbing compounds (911, 912).

Saccharomyces cerevisiae degraded captan to thiophosgene and di(thiophosgene) (910).

Captan has been shown to react with NaDDC, $Zn(DDC)_2$, and $Fe(DDC)_3$ (909).

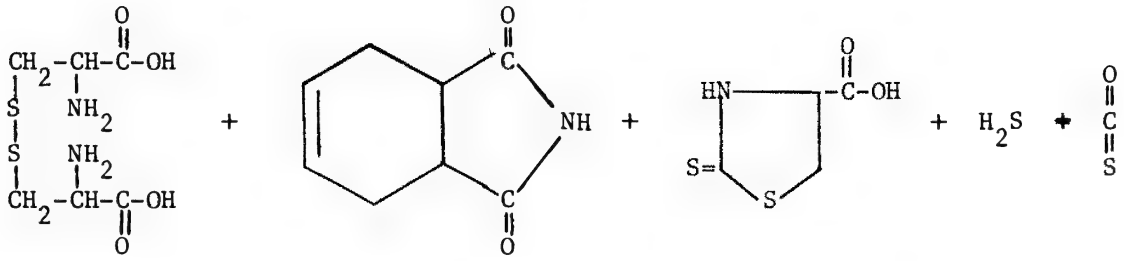
$$\text{Captan} + DDC^- \rightarrow \text{Bis(dimethylthiocarbamyl)trithiocarbonate}$$

$$TMTD + TMTM + Cl^- + COS$$

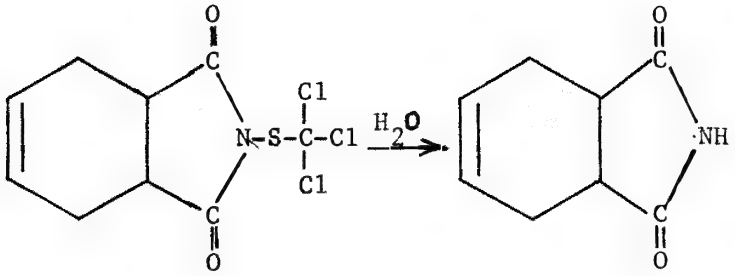


Captan

Cysteine



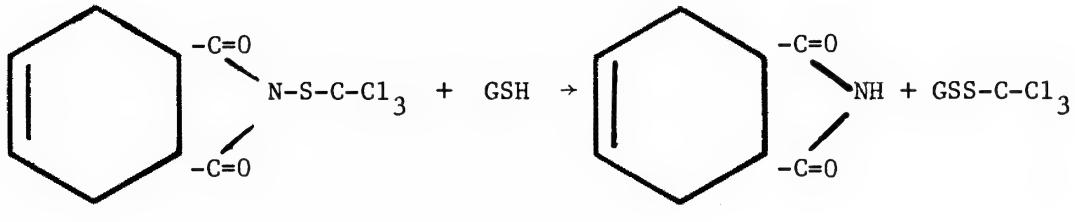
Cystine + Tetrahydro-Phthalimide + 2-Thiazolidine-thione-4-carboxylic Acid + H_2S + CSO

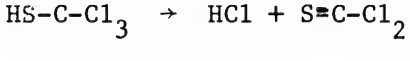


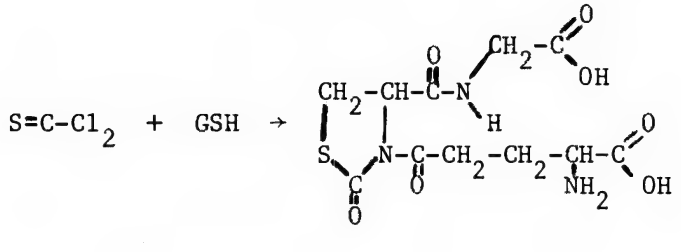
Di(thiophosgene)

CS_2 + H_2S

In other studies, after incubation of labeled captan with Neurospora crassa conidia, most of ^{35}S was found to be bound to water-soluble and protein fractions. ^{35}S occurred largely in oxidized glutathione and in a compound believed to be a thiazolidine derivative of glutathione. The same product was obtained by reacting glutathione with carbonyl sulfide, a known decomposition product in the reaction of captan with thiols (1208, 1689).







Carbaryl (Sevin) (1-Napthyl-N-methylcarbamate)

Labeled carbaryl was administered intraperitoneally in polyethylene glycol to guinea pigs and rats. Within 24 hours, 85% of the administered count was recovered in the urine in both cases (806).

When naphthyl labeled carbaryl was administered to the rat, $C^{14}O_2$ was not found. However, methyl and carbonyl labeled carbaryl gave rise after four days to residues amounting to 11 and 32% respectively, of the dose as $C^{14}O_2$. N-methylcarbamic acid rose by hydrolysis of the ester bond and then decomposed to CO_2 and methylamine. The latter was oxidatively demethylated to CO_2 and formate. Excretion of naphthyl and carbonyl labels was essentially complete with 95% and 99% of the label, respectively, recovered. The methyl label was excreted to the extent of 88% and residues were detected in the intestinal tract, carcass and organs (630, 806).

In the urine of rats in addition to 1-naphthol (IV) (226), 1-naphthyl methylcarbamate N-glucuronide (V), 1-naphthyl methylimido-carbonate O-glucuronide (VI), 4-(methylcarbamoyloxy)-1-naphthyl glucuronide (VII), 1-naphthyl glucuronide (IX), 1-naphthyl sulfate (X), 4-(methylcarbamoyloxy)-1-naphthyl sulfate (VIII), three unidentified compounds (806, 808, 836) and a compound believed to be 1-naphthyl N-hydroxymethylcarbamate (III) were observed (630). Similar results were observed with guinea pigs.

In dogs administered naphthyl-C^{14} and N-methyl-C^{14}, 35% and 11%, respectively, of the labels were excreted in feces; 40 and 23%, respectively, in urine over a seven day period. None of the major metabolites observed in urine of rats was observed with dogs. The dog can conjugate napthol and appears to conjugate carbaryl directly. A hydrolytic pathway involving the N-methyl group was indicated (809).

Rabbits, treated with carbaryl, excreted in the urine small amounts of about eleven metabolites, six of which were identified as compounds II, III, IV, XI, XII and XIII (857, 858).

After administration of C^{14}-labeled carbaryl to lactating cows, radioactive residues were observed in whole milk and milk fat, tissues, urine and feces. In addition to 1-naphthol in the urine, five compounds were observed in feces and milk: 5,6-dihydroxy-5,6-dihydronaphthyl N-methyl carbamate; 1,5,6-Trihydroxy-5,6-dihydronaphthalene; 1-naphthyl-N-hydroxy-methylcarbamate; 4-hydroxy-1-naphthyl N-methylcarbamate; and unchanged carbaryl (374, 1495, 1550).

Naphthyl-C^{14} and methyl-C^{14} labeled carbaryl was administered to pigs, sheep, monkeys and two men. The major metabolite observed in urine were VI and VII. In addition to these two metabolites, small amounts of compound IX was observed with pigs; VIII and IX, with sheep, VIII, with monkeys; and IX and X with the men (1577). In studies on the effects of carbaryl on a work population, engaged in its manufacture, workers excreted both free and conjugated napthol in the urine (110). It was also shown that human liver converted carbaryl to 1-naphthol and other water soluble compounds (944) After exposure of carbaryl to plasma of 15 different animals, 1-naphthol was identified in each case (242).

Rat livers were shown capable of degrading carbaryl to 1-naphthol and water soluble metabolites (944). Using rat liver microsomes, carbaryl was degraded into six known compounds and at least as many unidentified materials. The methyl group was modified to give 1-naphthyl-N-hydroxymethylcarbamate. Ring hydroxylation occurred to yield 4-hydroxy- and traces of 5-hydroxy-1-naphthyl-N-methylcarbamate and 5,6-dihydroxy-5,6-dihydro-1-naphthyl-N-methylcarbamate. There was some evidence that the 3,4-dihydroxy-3,4-dihydro-analog was also formed. In addition to these materials, the hydrolysis product 1-naphthol was also found. Water-soluble fractions, probably conjugates of carbamates and their hydrolytic products, were obtained but not identified. These would be expected to consist of glucosiduronic acids, sulfates, mercapturic acids, N-acetylmercapturic acids and glucosides (378, 379, 1111, 1112).

Bean plants were injected with labeled carbaryl. Six days later the water soluble metabolites were recovered and treated with β-glucosidase. The following aglycones were recovered: 5,6-dihydroxy-5,6-dihydrocarbaryl, N-hydroxymethyl carbaryl, 4- and 5-hydroxycarbaryl, and 1-naphthol (845, 846).

In cotton plants, carbaryl was readily absorbed by the root system. Hydrolytic and non-hydrolytic paths contributed almost equally to the metabolism of carbaryl. In the hydrolytic path, a specific carbamate esterase catalyzed the hydrolysis of the ester bond, liberating 1-naphthol and N-methylcarbamic acid. The latter underwent spontaneous degradation to give up CO_2. A small portion (about 3%) of the absorbed material was eliminated as a basic volatile material, probably unchanged amine. Using appropriately C^{14}-labeled carbaryl, it was shown that the methyl group underwent oxidation. For the non-hydrolytic path, there was evidence of oxidation of the intact carbaryl, probably to a hydroxylated metabolite (1045).

Carbaryl persisted longer in soil water than in lake water. Four days after treatment of soil water, 1-naphthol as well as carbaryl was recovered (872).

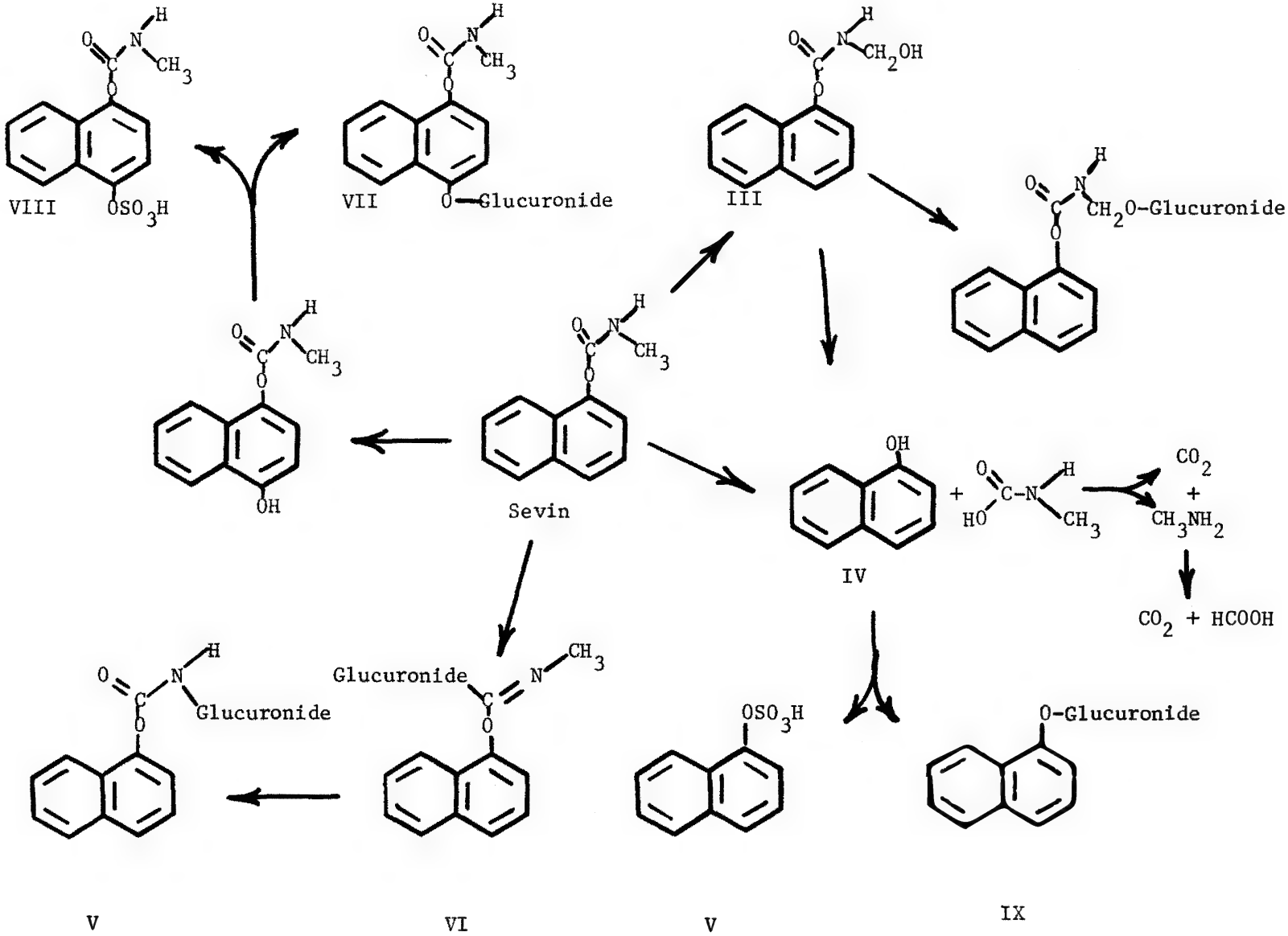
Topically applied solutions of carbaryl in acetone were absorbed fairly rapidly by milkweed bug nymphs. Only unchanged carbaryl and a very polar unidentified metabolite were identified. With the German cockroach, (Blatella germanica L.) absorption was slow and excretion rapid. 1-Naphthol was evident in the excreta at 24 hours and in tissues at 72 hours. Six other unidentified metabolites were demonstrated. Two were identified only as conjugates of 1-naphthol; one contained sulfur, probably as sulfate, and one contained a carbohydrate tentatively identified as glucose (399, 841). In the housefly, three metabolites were demonstrated. Changes undergone by 1-naphthol showed that it formed the same series of more polar products, indicating hydrolysis of the ester bond as the first step in the metabolism of carbaryl (399). In the presence of fly homogenates, hydroxylation of carbaryl occurred at the 4- or 5- position and the N-hydroxymethyl analog was also formed (1427, 1779).

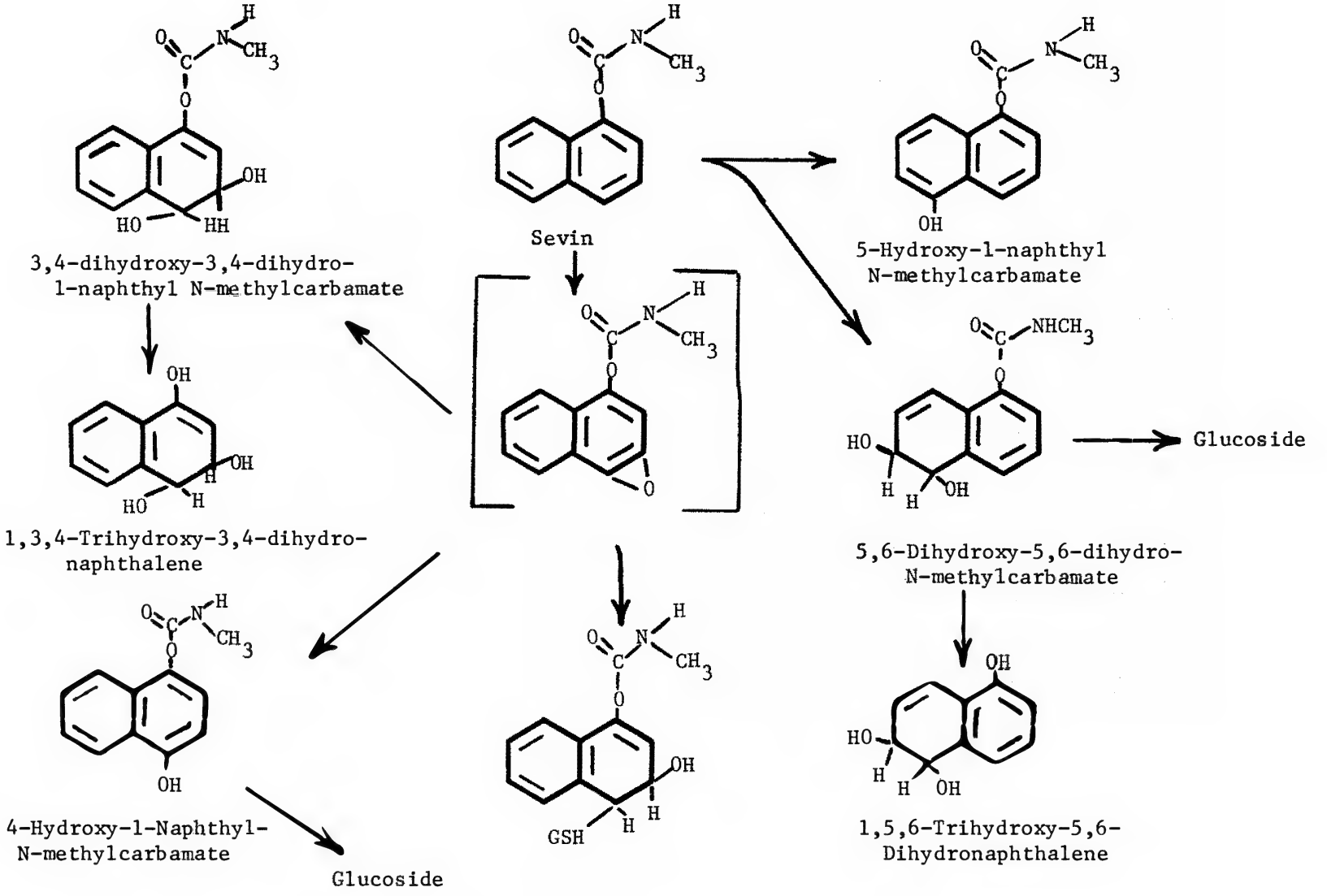
Stable flies (Stomoxys calcitrans L.) and rice weevils (Sitophilus oryza L.) converted carbaryl to 1-naphthol and four unidentified metabolites (224). Metabolites produced by Pseudomonas melophthora were not identified (145).

Adult boll weevils (Anthonomus grandis Boheman) were treated topically with labeled carbaryl. Subsequently, 1-naphthol, 1-naphthy-N-hydroxymethyl-carbamate, 5,6-dihydroxy-5,6-dihydro-1-naphthyl-N-methylcarbamate, and an unidentified compound were detected. 1-Naphthol increased markedly coincident with the rapid disappearance of 1-naphthyl-N-hydroxymethylcarbamate (25). Following injection of carbaryl into bollworm adults (Heliothus zea Boddie), 1,5,6-trihydroxy-5,6-dihydronaphthalene was observed in addition to those compounds found in the boll weevils. Metabolism of carbaryl by 5th-instar bollworm larvae was qualitatively similar to that of boll weevils but quantitative differences were observed (25).

The low toxicity of carbaryl to larvae of the cotton leaf worm (Prodenia litura F.) was attributed to the presence of enzyme systems capable of degrading this material: carbamate esterase, amine oxidase and catalase (631). Detoxication of labeled carbaryl in adult larva of the cotton leaf worm was primarily via non-hydrolytic paths involving hydroxylation. The hydrolytic path (about 14%) gave rise to 1-naphthol and N-methylcarbamic acid. The latter gave methylamine by decarboxylation, which then underwent oxidative demethylation to formate and CO_2 (1538).

1-Naphthol and phthalic acid have been identified by gas chromatography and thin-layer chromatography after ultraviolet exposure of carbaryl. Several other compounds were observed but not identified (3, 315, 1105, 391).

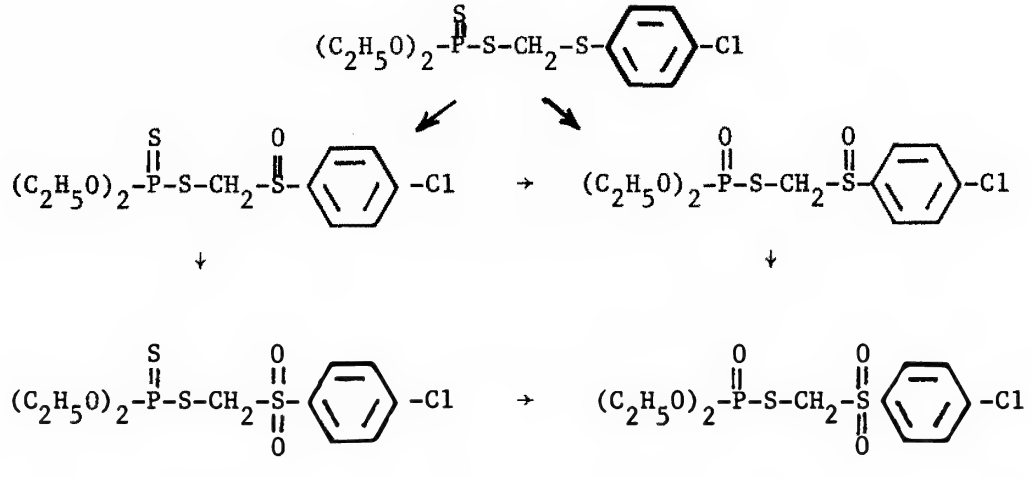

VIII
VII
III
Sevin
IV
V
VI
V
IX
Glucuronide
O-Glucuronide
CH_2O-Glucuronide
CO_2 + HCOOH


3,4-dihydroxy-3,4-dihydro-
1-naphthyl N-methylcarbamate
Sevin
5-Hydroxy-1-naphthyl
N-methylcarbamate
1,3,4-Trihydroxy-3,4-dihydro-
naphthalene
5,6-Dihydroxy-5,6-dihydro-
N-methylcarbamate
Glucoside
4-Hydroxy-1-Naphthyl-
N-methylcarbamate
Glucoside
1,5,6-Trihydroxy-5,6-
Dihydronaphthalene

Carbophenothion (Trithion) [0,0-Diethyl S-(p-chlorophenylthio)methyl phosphorodithioate]

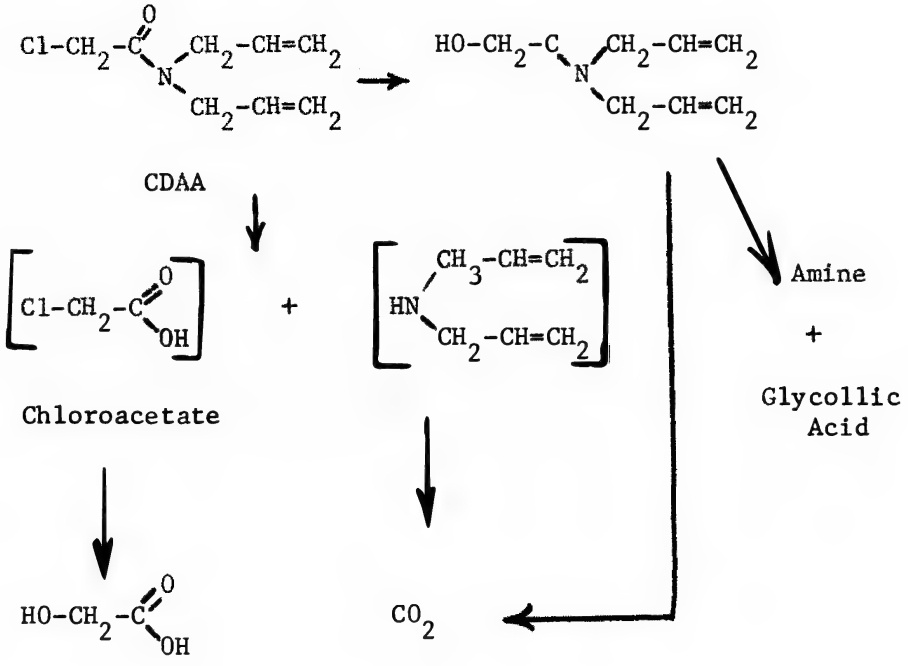
The oxidative metabolism of trithion was studied on field-growing lettuce. Samples were removed at various times after spraying and analyzed. Results indicated that the principal route of oxidation involved thioether oxidation to form the sulfoxide and then the sulfone. This was followed by phosphorothionate oxidation to form the thiolphosphate sulfone (289).

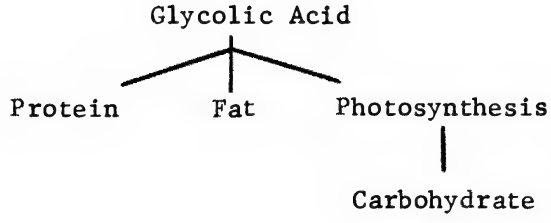
After exposure of carbophenthion to ultra-violet (maximum at 2540 A), the corresponding sulfoxide was observed (1608).



CDAA (α-Chloro-N,N-diallylacetamide)

Plant metabolism studies of CDAA-C^{14} indicated that this compound was degraded into CO_2 and glycollic acid with subsequent incorporation of the label in all fractions (725, 1303). CDAA also reacted with sensitive sulfhydryl groups (610, 724). In the soil, hydrolysis could also occur at the α-chlorine (610).



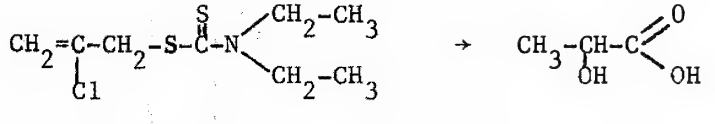


CDEC (Vegedex) (2-Chloroallyl-N,N-diethyldithiocarbamate)

Plants are able to metabolize the 2-carbon of the allyl group as $C^{14}O_2$. One of the primary metabolites of the 2-chloroallyl moiety was lactic acid. Distribution of radioactivity, as shown by using 2 dimensional chromatography, indicated that almost all amino acids were labeled with C^{14}, with the highest activity in asparagine, histidine, and glutamine (610, 725).

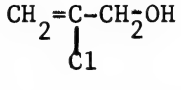
Irradiation of aqueous dispersions of CDEC with UV (2537 A) caused losses related to the length of exposure. Decomposition products were not identified (1393).

In soil, hydrolysis and oxidation can occur separately or concurrently (610).



CDEC

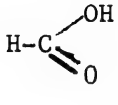
Lactic Acid



2-Chloroallyl Alcohol

Amino Acids

or



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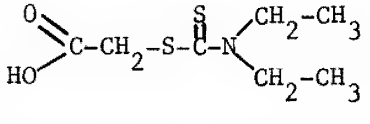
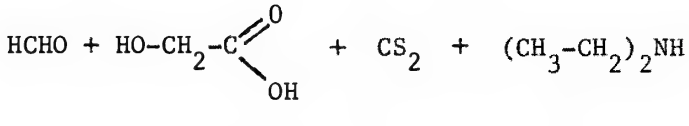


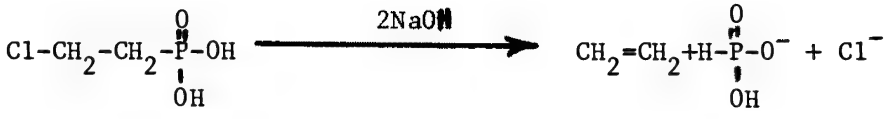
2-(diethylthiocarbamyl)acetic acid



Glycolic Acid

CEPA (Amchem 66-329) [2-Chloroethylphosphonic Acid]

Plants (Bryophyllum cruentum Baker) were treated with CEPA. The breakdown of CEPA into ethylene was observed. In the presence of a base, this breakdown appears to be second-order and apparently leads to the production of phosphonate and chloride (1750).



CEPA Ethylene

Chlorothion [0,0-Dimethyl 0-(3-chloro-4-nitrophenyl) phosphorothioate]

Chlorothion was exposed to microsomes of rabbit and rat livers and of houseflies. The corresponding P=0 analog was observed only in the presence of $NADPH_2 + O_2$. Some conversion of the P=0 analog to the corresponding phenol was also observed (1700). Rabbit sera also degraded chlorothion to the phenolic analog (1078).

CEPC [2-Chloroethyl-N-(3-chlorophenyl) carbamate]
CIPC (Chloropropham) [Isopropyl N-(3-chlorophenyl) carbamate]
IPC (Propham) [Isopropyl N-phenylcarbamate]

When applied to plants, IPC was metabolized to the corresponding N-OH analog (91). An amidohydrolase was isolated from soybeans which hydrolyzed IPC (561).

In recent studies a bacterium, Pseudomonas sp., was found in soil that hydrolyzed phenylcarbamate herbicides to the corresponding aniline. While this is a general reaction for the phenylcarbamate esters, urea analogs were not hydrolyzed (773, 774, 775).

Pseudomonas striata, a flavobacterium, an agrobacterium, and an achromobacter were able to degrade CIPC, as demonstrated by production of 3-chloroaniline and subsequent liberation of chloride. An achromobacter and an arthrobacter were able to degrade CEPC (768).

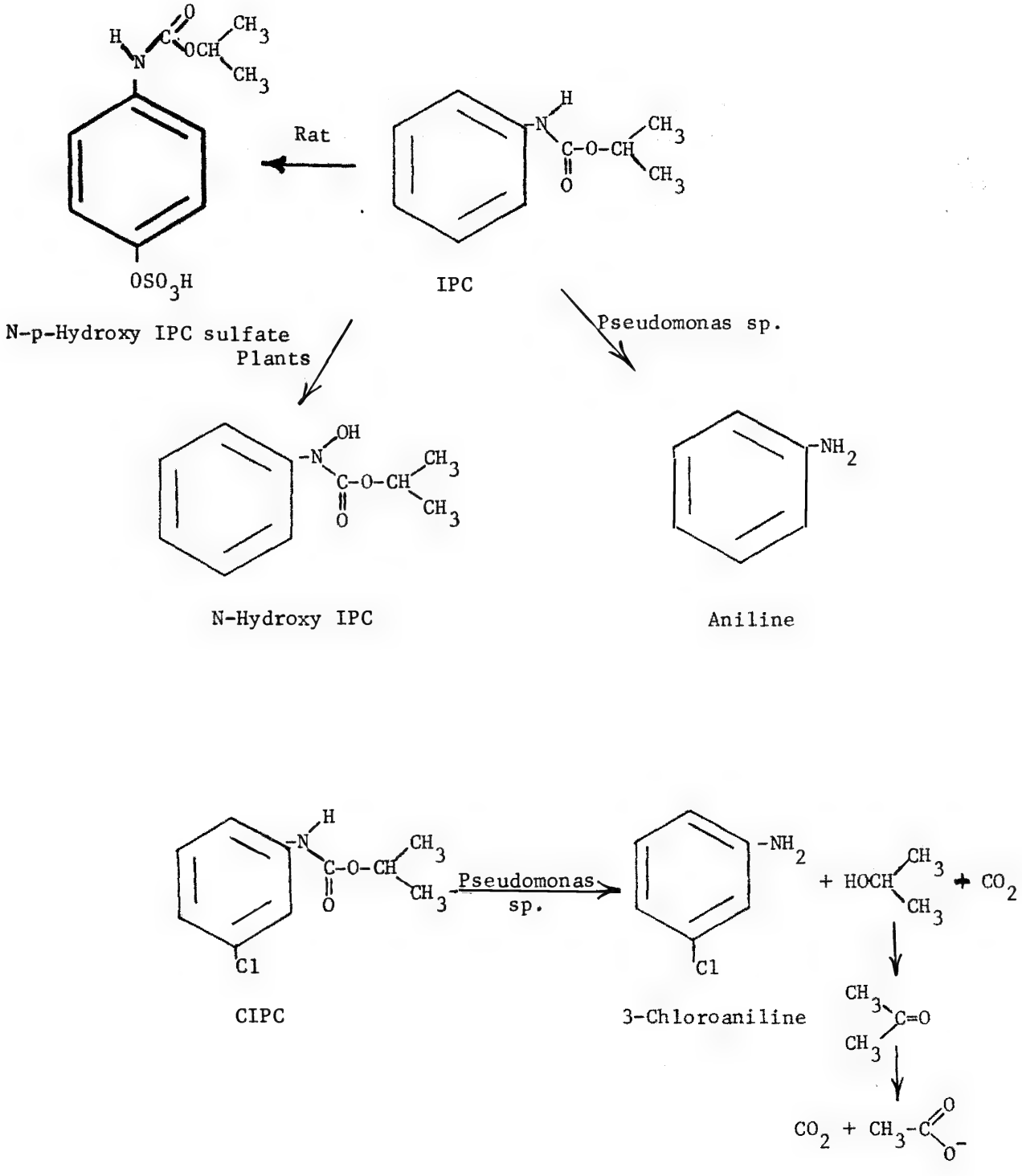
First order rate constants in terms of reciprocal time and heat of activation (H_a) were derived for IPC and CIPC (211).

	$T^{o}C$	k (day^{-1})	H_a
IPC	15	4.67×10^{-2}	7,768
	29	8.79×10^{-2}	
CIPC	15	4.25×10^{-3}	21,247
	29	2.39×10^{-2}	

In other studies conducted with microorganisms derived from the activated sludge and effluent of a sedimentation basin, CIPC was hydrolyzed and metabolized. The phenyl ring was attacked and degraded. Metabolism via 4-chlorocatechol and the ketoadipic acid system was proposed (1594).

(SEE ALSO UC-10854).

In recent studies, rats were administered IPC intraperitoneally. Examination of collected urine, showed the presence of isopropyl N-p-hydroxyphenylcarbamate as the O-sulfate (1688).


Rat
IPC
N-p-Hydroxy IPC sulfate
Plants
Pseudomonas sp.
N-Hydroxy IPC
Aniline
CIPC
Pseudomonas sp.
3-Chloroaniline

Chloranil

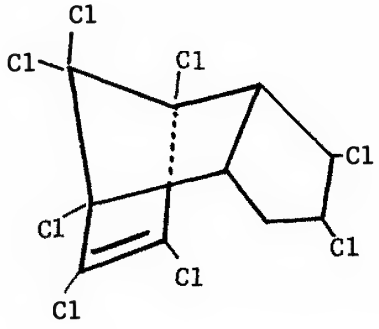
Studies showed that exposure of chloranil to ultra violet light resulted in a two step reduction (1547). When chloranil was added to cultures of Aspergillus niger, Neurospora crassa, or Mucor sp., a rapid buildup of free radicals was observed by electron spin resonance spectroscopy. Yeast suspensions and extracts also exhibit a rapid build-up and decay of a free radical. This corresponded to the semiquinone (1617).

Chlorate

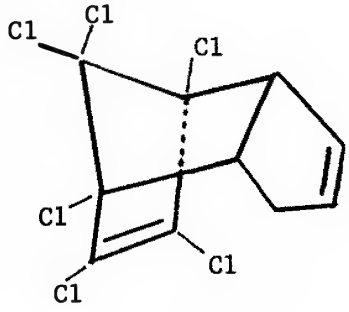
The toxicity of chlorate to wheat plants appeared to be coupled to a nitrate reductase system and to be caused by ClO_2^- that resulted from reduction of ClO_3^- by this system (874).

Chlordane (Chlordan) (1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene)

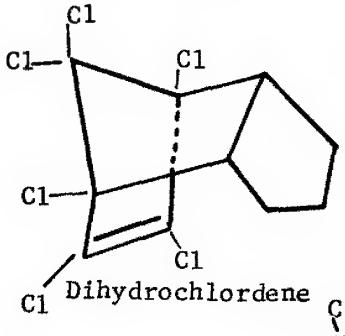
Studies with Wistar rats that received an intravenous dose of α-chlordan-C^{14} showed that 29% of the total injected radioactivity was excreted within 60 hours in feces and that only 1% was excreted in urine. Analyses showed that 75% of the radioactivity found in the feces were hydrophilic metabolites (1173).



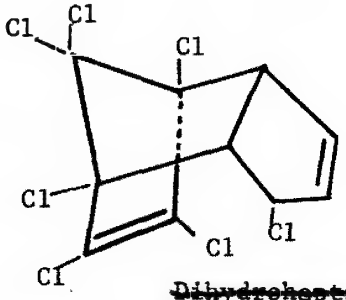
Chlordane



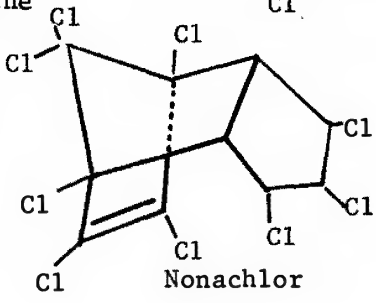
Chlordene



Dihydrochlordene



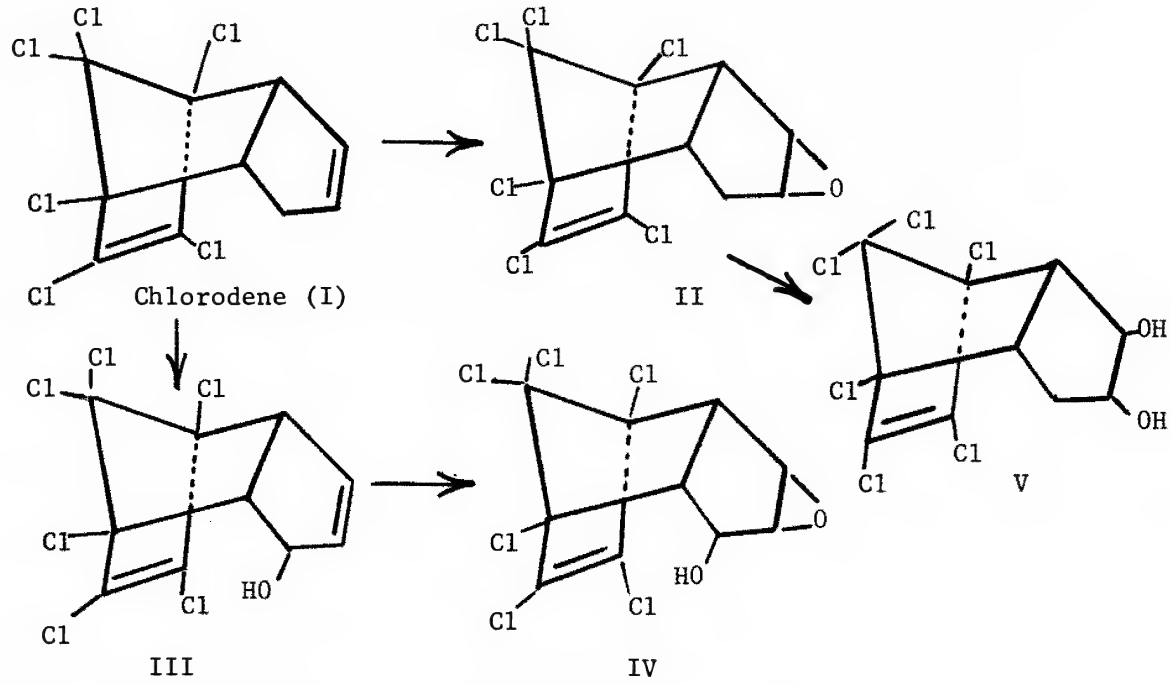
~~Dihydroheptachlor~~ Heptachlor



Nonachlor

Chlordene (4,5,6,7,8,8-Hexachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene)

Houseflies converted chlordene into an epoxide (II) and two compounds identified as 1-hydroxychlordene (III) and the 1-hydroxy epoxide (IV). Chlordene also underwent atmospheric oxidation. Compounds (II) and (III) were identified in the resultant mixture of products (187, 191). When incubated with pig liver microsomes, chlordene epoxide was converted into a diol (V) (1746).



1-Bromochlordene

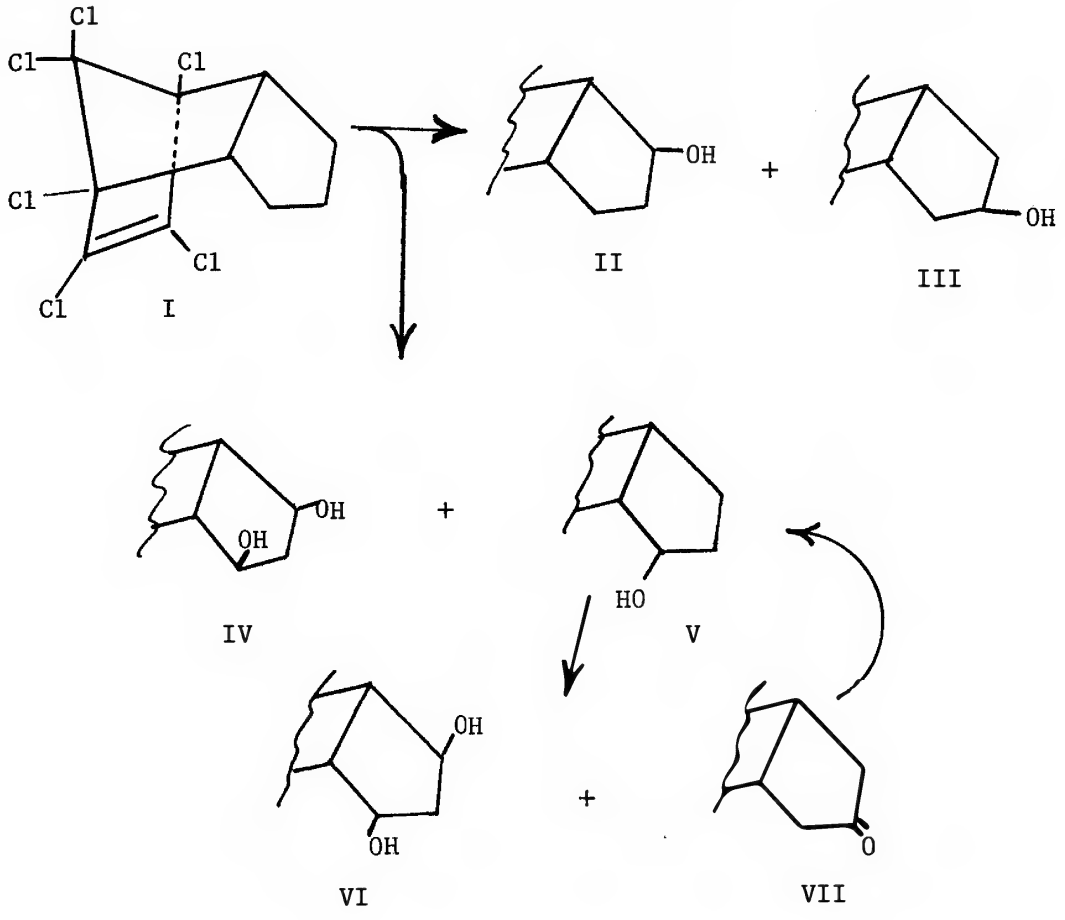
Corn borers metabolized this compound to the corresponding epoxide (1058).

Dihydrochlordene (4,5,6,7,8,8-hexachloro-1,2,3a,4,7,7a-hexahydro-4,7-methanoindene)

Dihydrochlordene (I) was incubated with housefly and pig liver microsomes, nicotinamide, and NADP. Evidence indicated that two of the metabolites were (II) and (III). Traces of IV and V were believed to be present also.

Incubation of (II) with pig liver or housefly microsomes and $NADPH_2$ gave rise to a diol, identical to that produced by hydroxylation of dihydrochlordene and believed to be IV.

After incubation of Compound V with pig liver or housefly microsomes and $NADPH_2$, the trans-diol VI was formed. Another compound, believed to be 2-ketodihydrochlordene, (VII) was also found. Incubation of the latter with added $NADPH_2$ gave rise to V (189).



Dihydroheptachlor (DHC) (3,4,5,6,7,8,8-heptachloro-1,2,3a,4,7,7a-hexachloro-4,7-methanoindene)

α-DHC

On gas chromatographic columns, α-dihydroheptachlor (α-DHC) underwent thermal decomposition to chlordene. Incubation of α-DHC with pig liver or housefly microsomes and $NADPH_2$ gave rise to two unidentified compounds believed to be chlorohydrins (190).

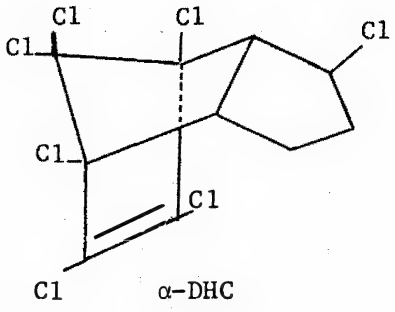
β-DHC

Gas chromatography of β-DHC caused some thermal decomposition to chlordene. Incubation of β-DHC with pig liver microsomes, in the absence or presence of $NADPH_2$, gave rise only to a small amount of chlordene. In the presence of $NADPH_2$ and housefly or pig liver microsomes, β-DHC gave rise to chlordene, chlorhydrin (IV), a diol (V), 2-ketodihydrochlordene (VI) and two unidentified compounds (190).

β-Dihydroheptachlor was also metabolized by Aspergillus niger and Penicillium notatum to more hydrophilic compounds. However, none were identified (1174)

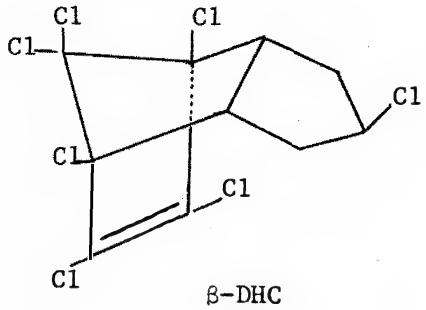
γ-DHC

In the absence of $NADPH_2$, pig liver microsomes gave rise only to small amounts of chlordene. In the presence of $NADPH_2$ and housefly or pig liver microsomes, γ-DHC gave rise to an unidentified chlorinated hydrocarbon plus two alcohols (VIII & IX), 2-ketodihydrochlordene (VI) and a chlorohydrin (VII). (It was believed that compound VII gave rise to V.) With pig liver microsomes and $NADPH_2$, γ-DHC also gave rise to the trans diol (V) (190).



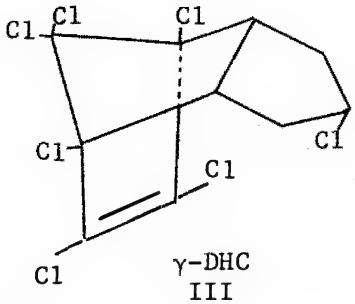
α-DHC

I



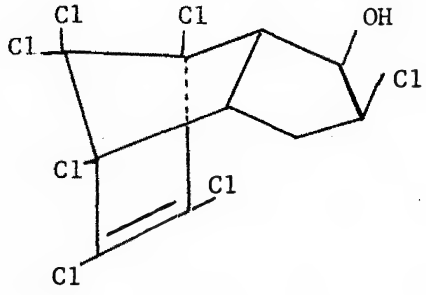
β-DHC

II

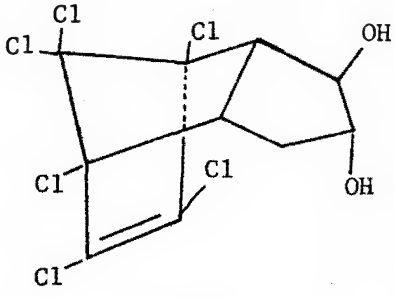


γ-DHC

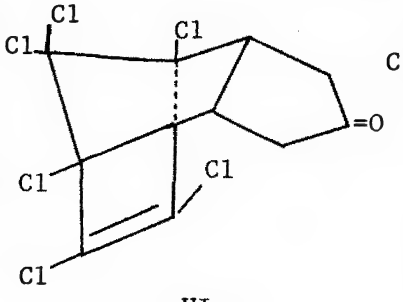
III



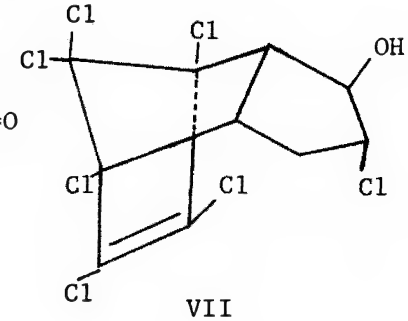
IV



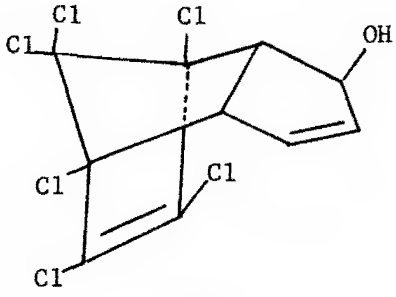
V



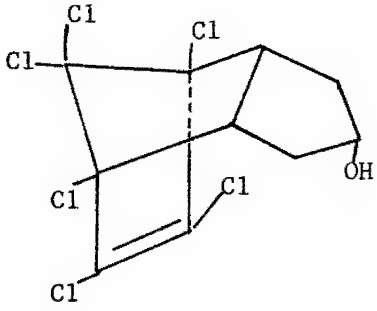
VI



VII



VIII



IX

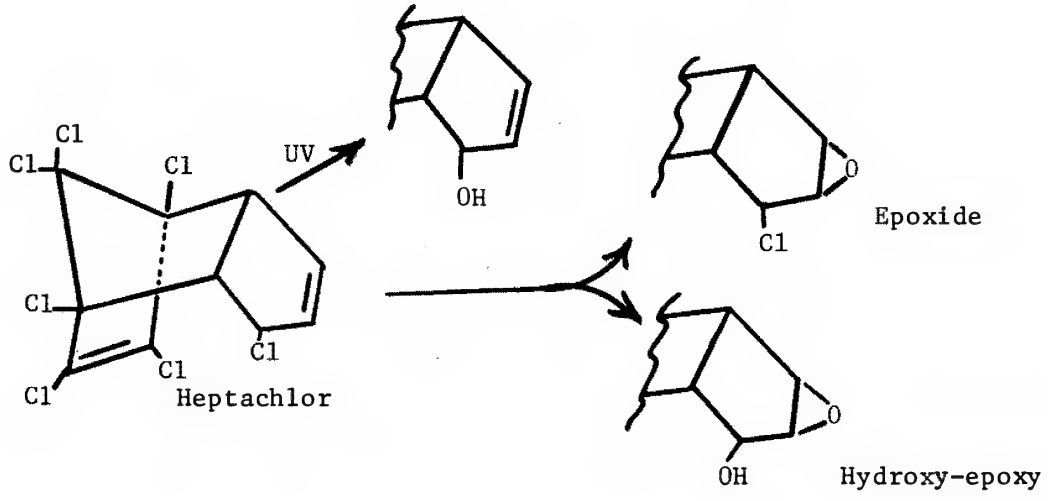
Heptachlor (1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene)

When heptachlor was fed to rats (1187), dogs (345), and cows (346) or when cows grazed on heptachlor treated pasture (64, 404, 1254, 1255, 1366), only heptachlor epoxide was found in the tissues of these animals and in the cows' milk. Studies have shown that epoxidation of heptachlor to heptachlor epoxide occurred in the microsomal fraction of the liver in rats and rabbits (1069, 1521).

After intravenous injection into rats and rabbits, heptachlor was metabolized to the epoxide and a hydrophyllic compound. In male rabbits, this amounted to 20% and 80%, respectively, for the two compounds within 144 hours after injection. By means of thin-layer, paper and gas chromatography, the hydrophyllic material was identified as 4,5,6,7,8,8-hexachloro-1-exo-hydroxy-6,7-exo-epoxy-1,2,3a,4,7,7a-hexahydro-1,4-endomethylenindene (802).

Heptachlor was rapidly metabolized by houseflies to heptachlor epoxide (1153). No other metabolite was found. Similarly, the only metabolite recovered from plants (522) or from soil (90, 521, 1059, 1500, 1531) was heptachlor epoxide.

In aqueous suspensions of heptachlor exposed to UV, 1-hydroxy-chlordene has been found (152). After UV exposure of hexane solutions of heptachlor, six unidentified degradation products were observed. Heptachlor epoxide in hexane gave rise to one compound (78).



Chlorfenvinphos (Supona, SD 7859, GC-4072, Birlane) [2-Chloro-1-(2,4-dichlorophenyl) vinyl diethyl phosphate]

Peak concentration of radioactive material in the urine of steers, after a spray application of P^{32}-labeled chlorfenvinphos, occurred in samples collected at three hours post-treatment. Metabolism resulted in the elimination of 9 or 10 unidentified radioactive compounds in the urine (266).

Residues of chlorfenvinphos and trichloroacetophenone were below limits of detection in organs of exposed sheep. Residue levels never reached 0.1 ppm (1224).

After oral administration to rats and dogs, C^{14}-chlorfenvinphos was rapidly metabolized and excreted. About 90% was eliminated in the urine; the remainder, via feces and, to a lesser extent, via expired gases. In rats, 32.3% of the dose was accounted for by 2-chloro-1-(2,4-dichlorophenyl) vinyl monoethyl phosphate (II); 41%, by 1-(2',4'-dichlorophenyl) ethyl β-D-glucopyranosiduronic acid (VI); 7%, by 2,4-dichloromandelic acid (XI); 2.6%, by 2,4-dichlorophenylethanediol glucuronide (X); and 4.3%, by 2,4-dichlorohippuric acid (IX). In dogs this was 69.6% (II); 3.6% (VI); 13.4% (VII), 2.7% (X) (704).

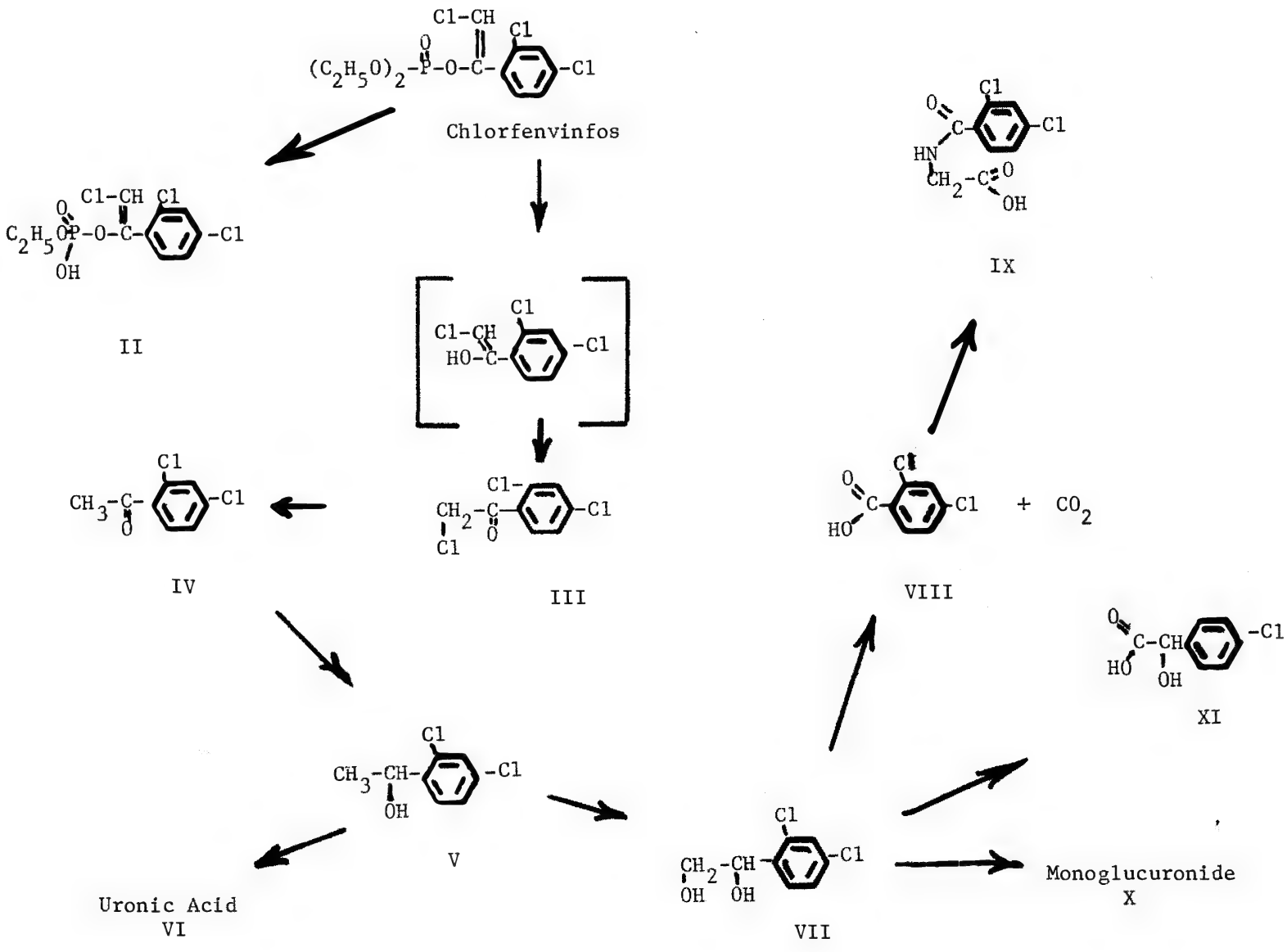
After intravenous administration, chlorfenvinphos was rapidly metabolized to the O-desethyl analog (II) (83%), compound XI (9%), and compound VI (8%) (704) in dogs.

Metabolism of the intermediate metabolites was also studied. After oral administration of compound II to a dog, XI (40%), VI (50%) and unchanged II (10%) were found in the urine. Following oral administration of labeled 2-chloro-1-(2,4-dichlorophenyl) ethanol, 80% of the label appeared as the corresponding glucuronide and 20% as compound XI. Since the glucuronide did not occur among the metabolic products of chlorfenvinphos, it is unlikely that the ethanol compound is a metabolite of the insecticide, at least in dogs (704).

Orally administered 2,4-dichlorophenacyl chloride (III) was excreted almost quantitatively by rats via the kidneys in urine as XI (30%), IX (5%), and VI (60%) (704).

Recent studies have shown that this compound reacted with and was bound by proteins in the blood plasma of mammals and in homogenates of house fly heads (155).

In Animals

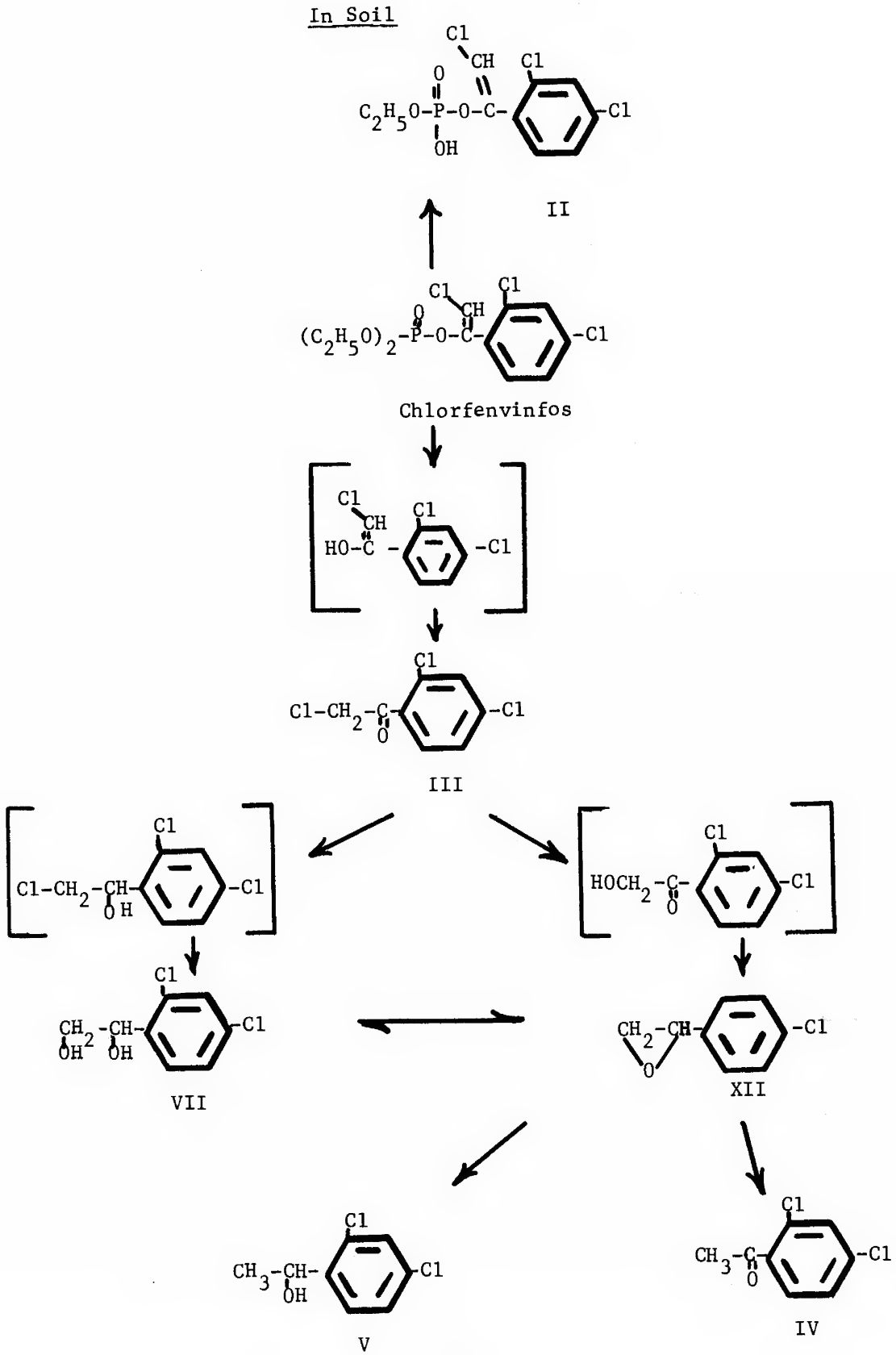

Chlorfenvinfos
II
III
IV
V
VI
VII
VIII
IX
X
XI
Uronic Acid
Monoglucuronide
+ CO2

Cabbage, grown to maturity in soil treated with chlorfenvinphos did not contain detectable residues of the parent compound or its breakdown products. Under the same conditions, carrots contained residues of unchanged chlorfenvinphos, a salt or conjugate of desethyl chlorfenvinphos, and traces of 2,4-dichloroacetophenone. Onion bulbs contained only chlorfenvinphos and the salt or conjugate of desethyl chlorfenvinphos (115).

Chlorfenvinphos (I) was applied to the foliage of potatoes, cabbage and maize. Half was absent after 2 to 3 days. On cabbage there was some evidence of conversion of the trans (β) isomer to the cis (α) isomer. Compound V, the major metabolite as a sugar conjugate, and traces of desethyl chlorfenvinphos (II) were detected. Treated carrots contained traces of 2,4-dichloroacetophenone (IV) (116).

Four different soils were treated with C^{14}-chlorfenvinphos and stored at 22° for four months. At that time, the soils were extracted. Thin-layer and gas chromatography detected the following metabolites: (II) desethyl chlorfenvinphos; (VII) 2,4-dichlorophenylethan-1,2-diol; (V) 1-(2,4-dichlorophenyl)ethan-1-ol; (IV) 2,4-dichloroacetophenone; (III) 2,4-dichlorophenacyl chloride; and (XII) 2,4-dichlorophenyloxirane. An unidentified compound and salts or conjugates of desethyl chlorfenvinphos were also indicated (115, 116).

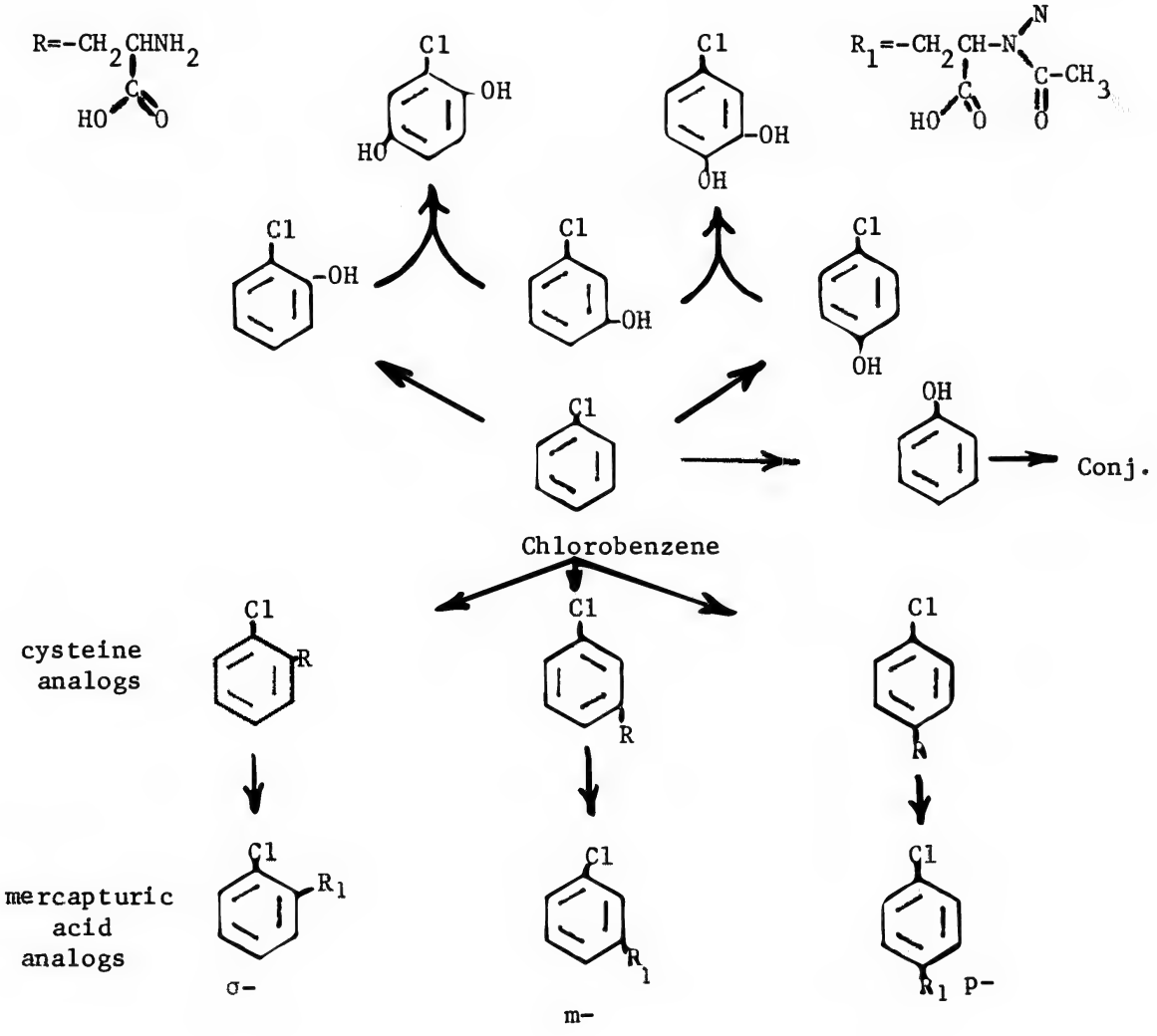
In peat, the residues of 2,4-dichlorophenacyl chloride built up to a maximum of 0.11 ppm at 15 weeks after application at 8 lb/acre. The half-life in soils varied from 2 to more than 23 weeks, depending on soil type, formulation, and the dosage level (113).


In Soil
II
Chlorfenvinfos
III
VII
XII
V
IV

Chlorobenzene

Locusts treated with chlorobenzene excreted o-, m-, and p-chlorophenol, 4-chlorocatechol and small amounts of 2-chloroquinol and phenol as O-conjugates (539).

Cysteine conjugates found in locust excreta were present as acid-labile precursors of chlorophenyl-L-cysteines and -mercapturic acids. Ortho -, m-, and p-chlorophenyl-L-cysteine and smaller amounts of m- and p-chlorophenylmercapturic acid were observed. An acylase present in crop fluid and excreta slowly hydrolyzed L-p-chlorophenylmercapturic acid to p-chlorophenyl-L-cysteine, which was enzymatically degraded to unidentified products (540).



2-Chloro-N-Isopropylacetanilide

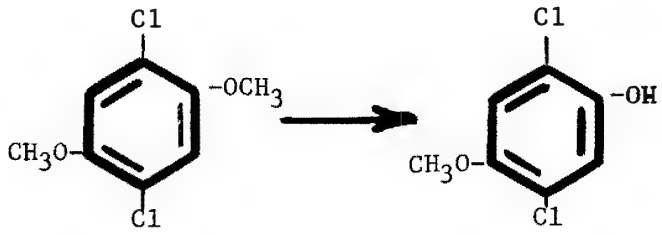
Ring tritiated 2-chloro-N-isopropylacetanilide was taken up rapidly by corn and soybean plants. Metabolism was rapid and no intact herbicide was detected in the seedlings five days after planting in treated soil. A water soluble metabolite containing a carboxy group and an N-isopropyl-aniline moiety was the main detoxification product (714, 1155).

N-(3-Chloro-4-methylphenyl)-2-methylpentamide

This compound prevented uptake of $C^{14}O_2$ but the mechanism or metabolism involved was unknown. Substituted anilides, in general, inhibit the Hill reaction and some have been shown to form lignin complexes (290).

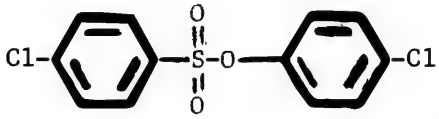
Chloroneb [1,4-Dichloro-2,5-dimethoxybenzene]

Mycelium of Rhizoctonia solani converted chloroneb to 2,5-dichloro-4-methoxyphenol. Chloroneb was metabolized by Neurospora crassa to an unidentified product (1120, 1778).



p-Chlorophenyl p-chlorobenzenesulfonate

S^{35}-labeled p-chlorophenyl p-chlorobenzenesulfonate was hardly decomposed in eggs and adults of the citrus red mite. When injected into the abdomen of the American cockroach, decomposition of this acaricide gave rise to p-chlorobenzenesulfonic acid. In citrus sapling and soybean seedling, the acaricide penetrated into the plant and was decomposed to p-chlorobenzenesulfonic acid and other compounds (1386).



p-Chlorophenyl p- chlorobenzenesulfonate

p-Chlorobenzenesulfonic acid

Ciba C- 9491 [O-(2,5-Dichloro-4-iodophenyl) O,O-dimethylphosphorothioate]

In treated sweet corn, traces of the O-analog and phenol were observed. The phenol also appeared to form a conjugate.(153).

Ciodrin (α-Methylbenzyl-3-hydroxycrotonate dimethyl phosphate)

A study of the absorption, elimination, and metabolism of orally administered p^{32} labeled ciodrin in a lactating ewe indicated moderate absorption and rapid elimination. Dimethyl phosphoric acid accounted for 61-90% of the radioactivity in the urine. Several other metabolites were noted but not identified. Similar results were obtained when the experiment was repeated with a lactating goat (255, 256).

Citicide (Polychlorinated turpentine)

When citicide was fed to rats, a portion passed through the alimentary canal unabsorbed and was excreted in the feces. In urine, very little citicide was excreted; however, chloride excretion was very high. Ethereal sulfate and glucuronic acid excretion was also significantly higher in citicide-fed animals than in controls. Mercapturic acid excretion of test animals was not significantly different than that of controls. Since halogenated aromatic hydrocarbons are detoxified in the liver via ethereal sulfate and glucuronic acid conjugates, it is quite probable that increased levels of these materials indicated detoxification processes of citicide (837).

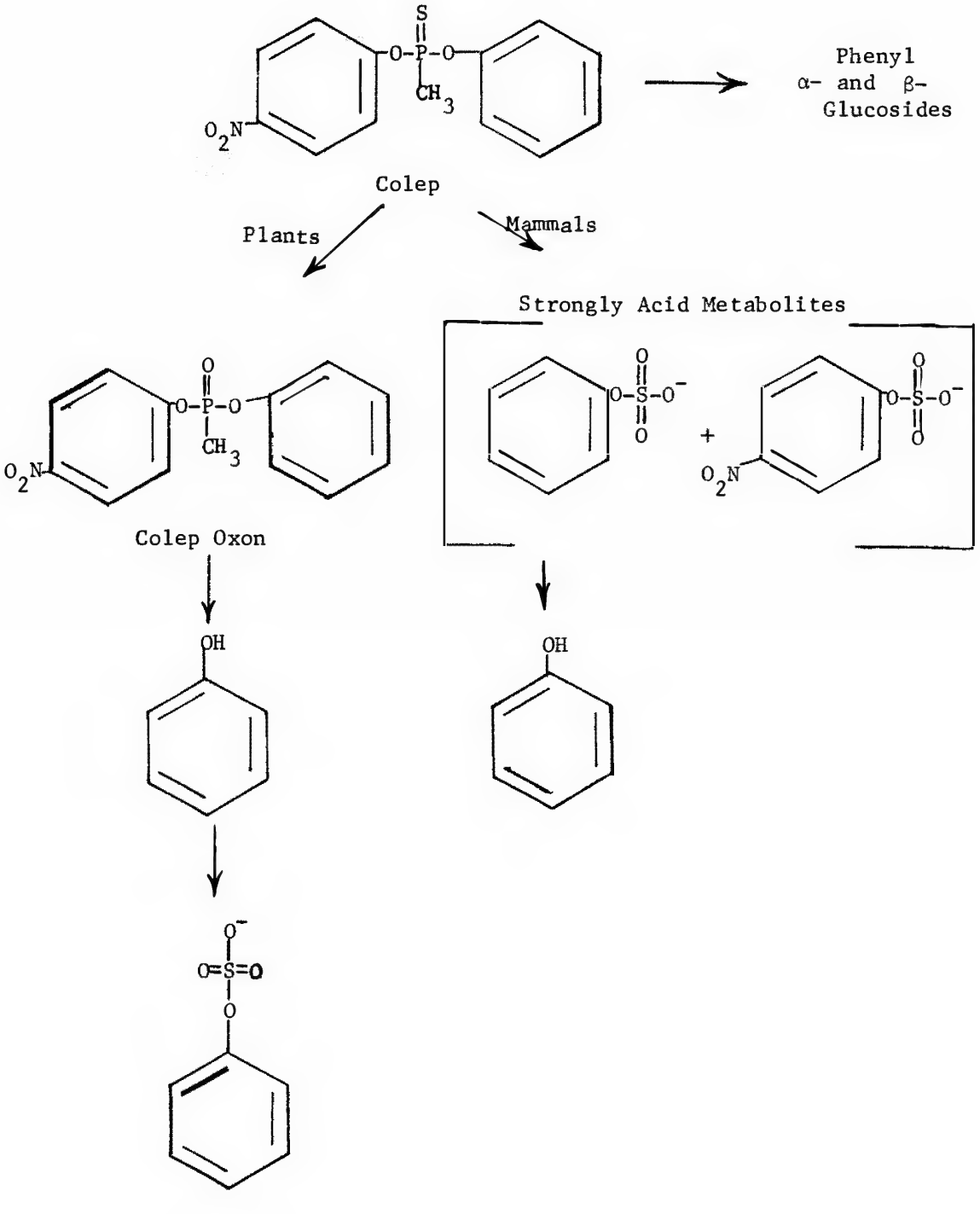
No citicide was found in the body fat and liver but chloride levels were higher than those for controls. This indicated the storage of metabolites in the body fat. None have been isolated thus far.

Colep [O-Phenyl-O'- (4-nitrophenyl) methylphosphonothionate]

The major portion of the radioactivity after administration of colep-C^{14} to rats was found in the urine within 24 hours. Liver and kidney contained little radioactivity after 24 hours, indicating that detoxification and excretion was nearly complete. No measurable activity was incorporated in the abdominal fat nor released as CO_2 (925).

The predominant excretory conjugate in mammals fed low levels of phenol was phenylsulfuric acid (1508). This type of compound was probably the strongly acidic metabolite found in the urine. After acid hydrolysis of urine samples, phenol was released.

After colep was applied to plants, the oxon analog was found in small amounts. The major amount of activity was apparently conjugated. Studies with acid and emulsin hydrolysis indicated the presence of phenyl β-glucoside and an α-glycoside. Another fraction contained phenyl sulfate. After acid hydrolysis of benzene, chloroform, and water extracts converted 75-80% of the radioactivity to phenol for both cotton and apple, thus showing the association of the radioactivity with phenol-containing materials (925).


Phenyl
α- and β-
Glucosides
Colep
Plants
Mammals
Strongly Acid Metabolites
Colep Oxon
OH
OH

Coumaphos (Coral, Bayer 21/199, muscatox) [0,0-Diethyl-0-(3-chloro-4-methylumbelliferone) phosphorothioate]

Dermal administration of Coumaphos-P^{32} to rats gave two peaks of radioactivity in the tissues. The first peak appeared between 4 and 6 hours following administration; the second, 4 to 7 days later. Two thirds of the excreted material appeared in the urine within 28 days, compared with four fifths within 18 days of subcutaneous and 14 days of oral administration (838, 877, 1100).

Four metabolites were detected in the urine. During the first 96 hours after oral treatment, diethyl phosphorothioic acid constituted 50 - 80% of excreted radioactivity; phosphoric acid, 10 - 20%; diethyl phosphoric acid, 10 - 20%; de-ethylated coumaphos and/or its oxygen analog, 5 - 30%. Following subcutaneous administration, only three metabolites were found. There was diethyl phosphorothioic acid but no phosphoric acid. After dermal treatment, none of the de-ethylated derivatives were found. Phosphoric, diethyl phosphoric, and diethyl phosphorothioic acids appeared in about equal proportions (877, 1100, 1446).

After subcutaneous administration, 17% of the radioactivity in the feces appeared to be coumaphos or its oxygen analog. Oral and dermal administration resulted in an increase to 50 - 60%. When the oxygen analog was orally administered, 10% appeared as diethyl phosphoric acid and 40% as the de-ethylated analog in the urine within 24 hours (1100).

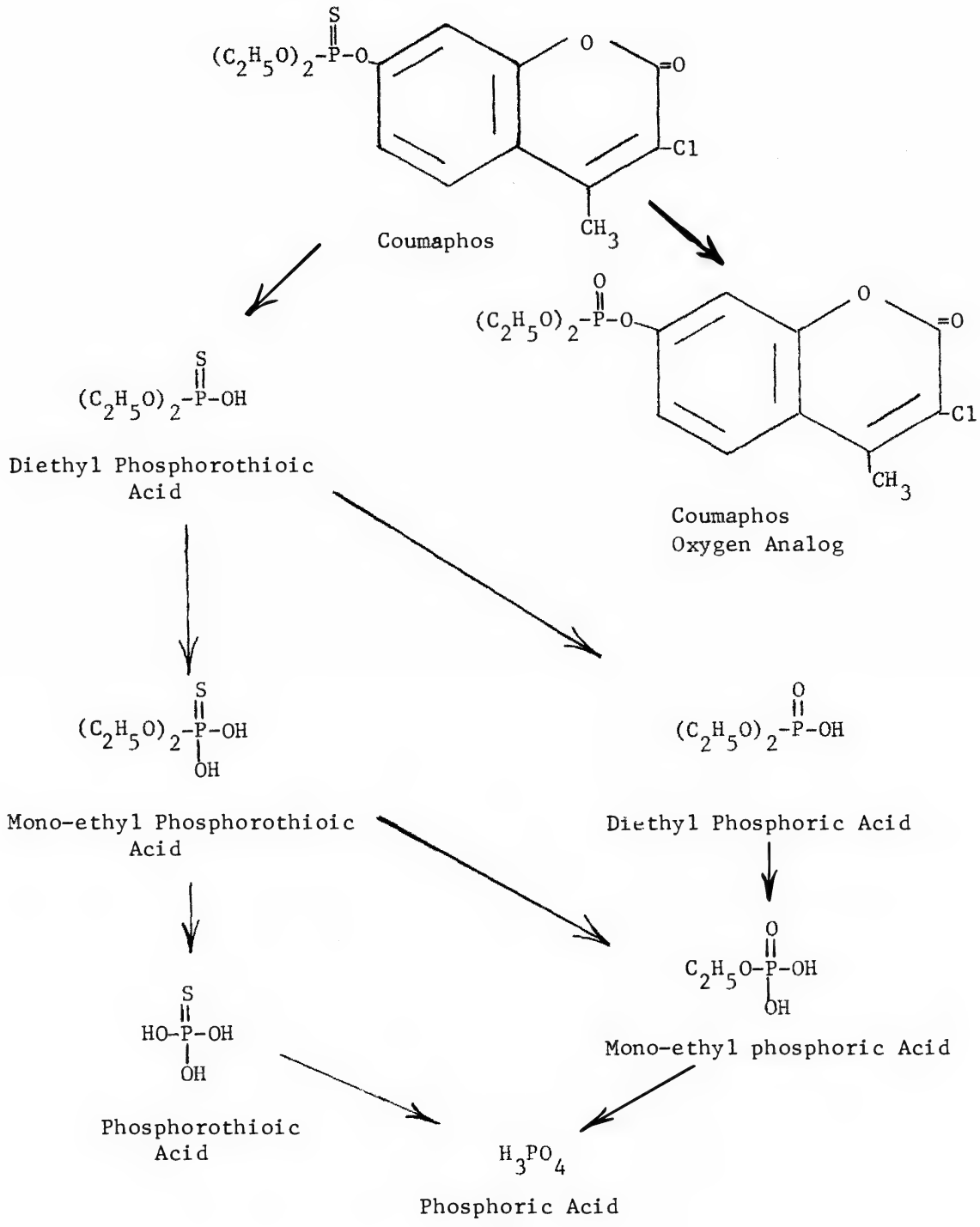
P^{32}-labeled coumaphos was applied dermally to a cow and a goat. Unextractable residues were significant in tissues of both animals. Hydrolysis products were below the limit of sensitivity of the method. Many tissues, particularly fat, contained considerable radioactivity that appeared to be the oxygen analog of coumaphos. The peak blood level of radioactivity for the cow occurred at 6 days; for the goat, at 6 hours and then again at 5 days. Cow cholinesterase continued to drop for seven days but recovered by the end of two weeks (757, 838, 1218).

Most of the radioactivity appearing in the urine was as hydrolysis products. Some of the oxygen derivative of coumaphos was found in the initial (up to 6 hours) collections but thereafter hydrolysis products were predominant: 35% phosphoric acid, 17% diethyl phosphoric acid, 29% diethyl phosphorothioic acid, and 18% as the de-ethylated coumaphos and its oxygen analog, primarily. Results obtained from the study with the goat were similar to those of the cow (757, 838, 1100, 1186).

When Rhode Island hens were fed mash containing labeled coumaphos, considerably more radioactive material appeared in the yolk than in the white or shell of eggs. The peak of radioactivity in the eggs appeared 11 to 15 days after the beginning of the treatment and was present as long as 6 to 8 days after return to normal diets. The decline in tissue residues

was not paralleled by an increase in egg residues. From feces both coumaphos and the oxygen analog, mono- and diethyl phosphoric acids, and diethyl phosphorothioic acid were isolated. There was evidence also that some coumaphos was completely degraded to phosphoric acid and subsequently incorporated into natural phosphorus containing metabolites (376, 377).

The cattle tick Boophilus microplus (Canestrini) was exposed to labeled coumaphos. Using liquid-liquid separation and paper chromatography, a number of compounds were found. Ultraviolet and infrared spectroscopy and chromatography were used to show the presence of unchanged coumaphos, the oxygen analog, ethyl phosphate, ethyl phosphorothioate, orthophosphate, desethyl analogs, and an unknown metabolite (1243).


Coumaphos
Coumaphos Oxygen Analog
Diethyl Phosphorothioic Acid
Mono-ethyl Phosphorothioic Acid
Diethyl Phosphoric Acid
Mono-ethyl phosphoric Acid
Phosphorothioic Acid
H_3PO_4
Phosphoric Acid

Coumarin (1,2-Benzopyrone)

A bacterium isolated from soil metabolized coumarin to L-tyrosine in the culture medium (865, 866). In other studies, coumarin was converted to 7-hydroxycoumarin by Penicillium (103). Additional studies have indicated that α-coumaric acid and melilotic acid were intermediates in the degradation of coumarin. When coumarin (I) was used as a substrate for Aspergillus niger, large amounts of melilotic acid (XX) were isolated. Small amounts of o-coumaric acid (XIV) and traces of 4-hydroxycoumarin (V) and catechol were also detected (129).

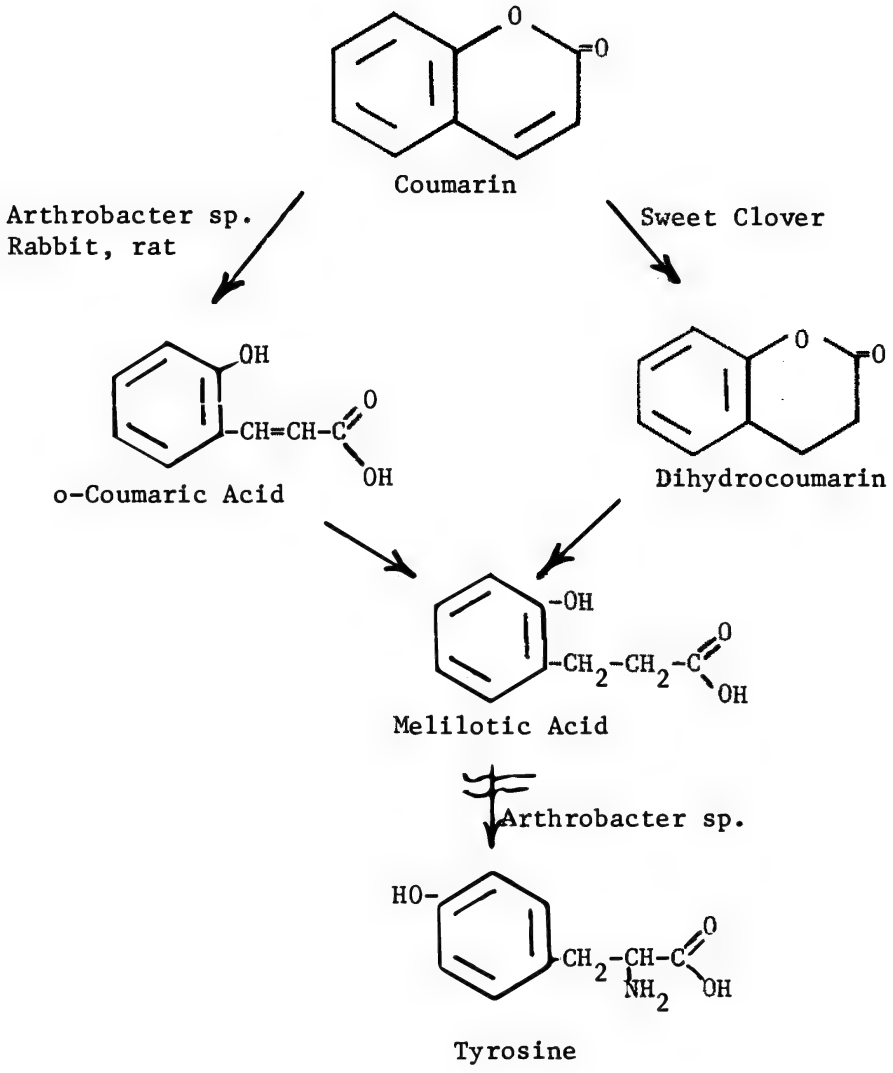
A similar pathway in animals was suggested by experiments with rats (140) and rabbits (140, 515, 516, 965). Urine from rabbits administered coumarin (I) was collected and examined for coumarin metabolites. 3-Hydroxycoumarin (II), 7-hydroxycoumarin (III), both free and as glucuronate conjugates and sulfate, and a trace of 8-hydroxycoumarin (XXIII) were detected. A small amount of o-coumaric acid (XIV) was detected in the free form and as the glucuronide. o-Coumaric acid was partially reduced, forming melilotic acid (XX), which cyclized to form dihydrocoumarin (XXII). Traces of o-coumaroylglycine (XIX) formed from o-coumaric acid (515, 965). When o-coumaric acid was administered to rabbits, melilotoylglycine (XXI), o-coumaroylglycine (XIX), umbelliferone (VIII), and 4-hydroxycoumarin (V) were detected in the urine (516). In other studies, o-hydroxyphenylhydracrylic acid (XV) o-hydroxyphenylacetic acid (XVII), and o-hydroxyphenyllactic acid (XVI) were also detected. Similar findings were obtained with rats (140). In addition to the 3-, 7-, and 8-hydroxycoumarins, which are excreted in conjugation, the ferret, guinea pig and mouse also excreted 5-hydroxycoumarin (XI) (965).

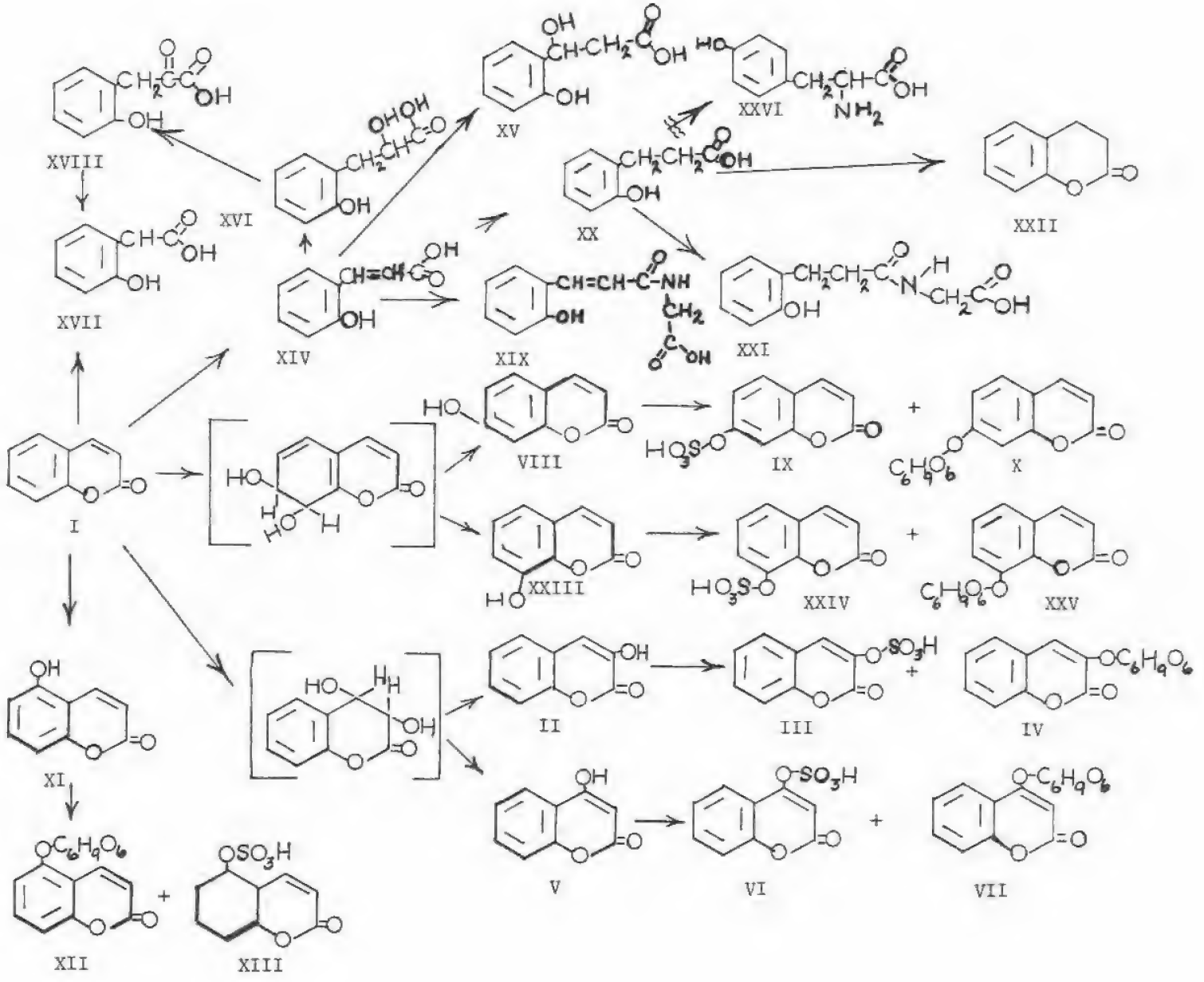
Metabolism of coumarin by liver microsomes of rats, guinea pigs, and rabbits was also studied. TLC, UV, and I.R. were used to identify metabolites: 3-HO coumarin (II), o-HO-phenyllactate (XVI), o-HO-phenylpyruvate (XVIII), and o-HO-phenylacetate (XVII) (454, 782).

In sweet clover, coumarin is rapidly converted to melilotic acid and its glucoside (832). It was postulated that coumarin was reduced to dihydrocoumarin and the pyrone ring was then opened to form melilotic acid. An enzyme was subsequently partially purified from sweet clover which catalyzed this ring opening (833). Exogenous coumarin was metabolized by lettuce seeds and seedlings. No metabolites were identified (1313).

In man, 7-hydroxycoumarin (III) (80%) and o-hydroxyphenylacetic acid (XVII) (6%) were found after coumarin ingestion (1789).

The fungus Aspergillus fumigatus Fresnius metabolized o-coumaric acid to 4-hydroxycoumarin and dicoumarol. From sequential induction, mass spectroscopy, co-chromatography and ultraviolet absorption spectra, it was deduced that 3-hydroxy-3-(2-hydroxyphenyl)propionic acid was an intermediate in the formation of 4-hydroxycoumarin. The latter was then converted non-enzymatically to dicoumarol (1660).




XVIII
XVII
XVI
XV
XXVI
XX
XXII
XIV
XIX
XXI
I
VIII
IX
X
XXIII
XXIV
XXV
XI
II
III
IV
V
VI
VII
XII
XIII

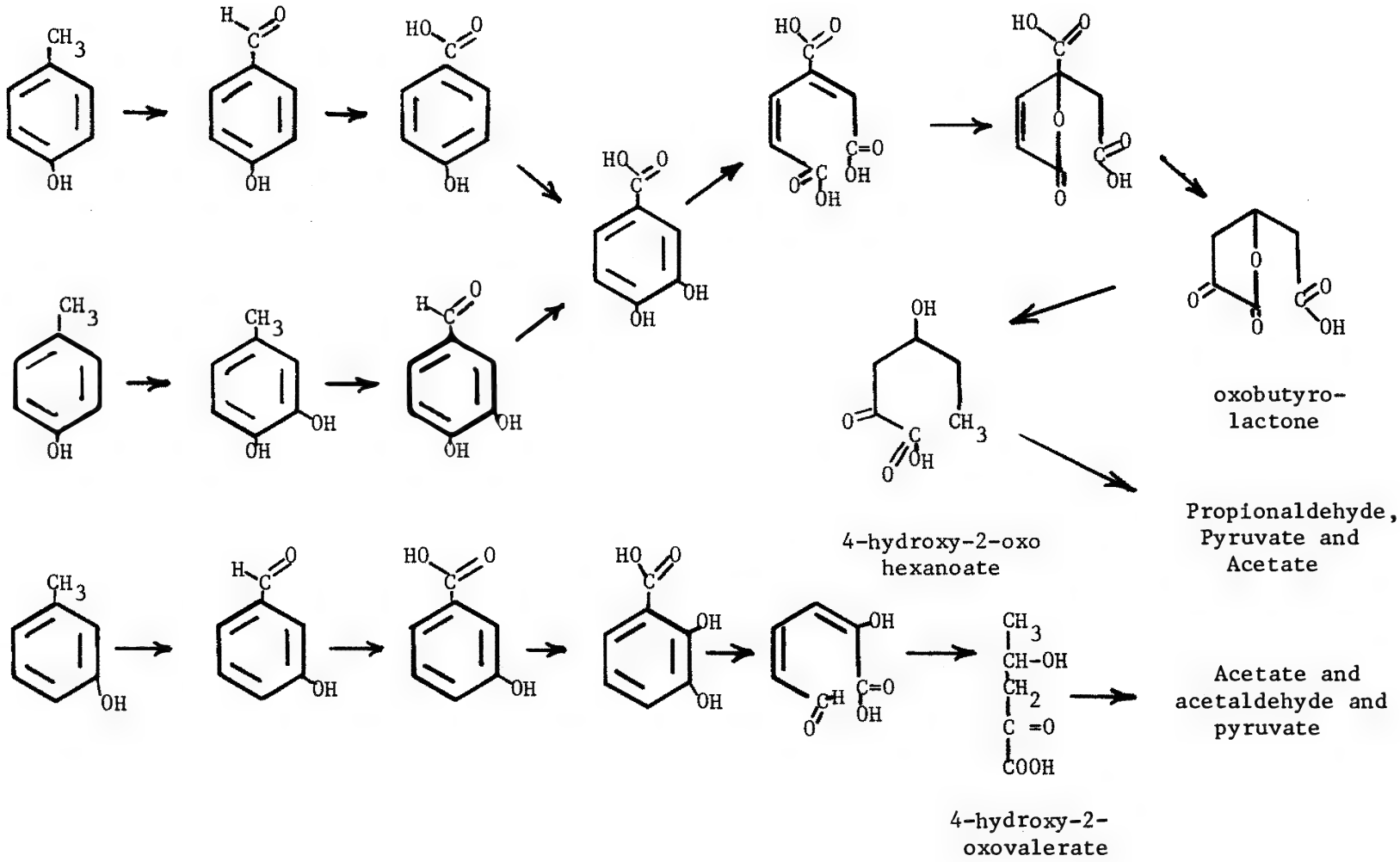
Cresols (Hydroxytoluene)

Pseudomonas N.C.I.B. 8893 initiated degradation of p-cresol by oxidizing the methyl group to carboxyl. Pseudomonas (U) left the methyl group intact and initially hydroxylated the benzene nucleus to give a catechol. Although specificity was exhibited in hydroxylation of cresols, the ring cleavage of the catechols appeared to be catalyzed by the same enzyme, catechol 2,3-oxygenase. Meta cresol and p-cresol were oxidized by Pseudomonas (U) to 3-methyl- and 4-methyl-catechol, respectively. Ring fission of 3-methylcatechol gave rise to 4-methyl-2-oxobutyrolactone, 4-hydroxy-2-oxovalerate, acetaldehyde, pyruvate, and acetate. Ring fission of 4-methylcatechol produced 4-ethyl-2-oxobutyrolactone, 4-hydroxy-2-oxohexanoate, propionaldehyde, pyruvate, and formate. Proposed metabolic pathways for cresols are shown (95, 329, 1607).

Cycloheximide (Actidione) [3-(2-(3,5-dimethyl-2-oxocyclohexyl)-2-hydroxyethyl) glutarimide]

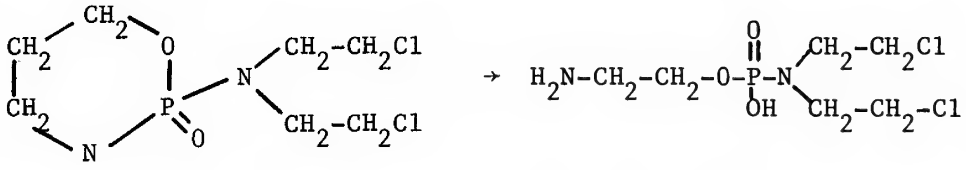
Bean plants were quite sensitive to cycloheximide and inactivation occurred rapidly, paralleling phytotoxicity symptoms. In cherry foliage, cycloheximide was apparently bound within the tissue (1348). When applied to tomato plants as the acetate, the free cycloheximide was formed and accumulated in the leaves (863).

CRESOLS


oxobutyro-
lactone
Propionaldehyde,
Pyruvate and
Acetate
4-hydroxy-2-oxo
hexanoate
Acetate and
acetaldehyde and
pyruvate
4-hydroxy-2-
oxovalerate

Cyclophosphamide [N,N-bis (2-chloroethyl)-N',O-propylenediamide]

In vivo activation on wheat root meristems occurred only when there was pretreatment with caffeine (1115).



Cycocel (CCC) [2-(Chloroethyl) Trimethylammonium Chloride]

Shoots of barley and chrysanthemums were placed for 24 hours in beakers containing labelled CCC. After removal, the shoots and residual solutions were tested for radioactivity. Paper chromatography indicated the presence of five radioactive products in both barley and chrysanthemum shoots. One compound was identified as choline. Additional assays showed that three of the compounds were metabolites of choline and indicated that the fifth compound was probably an intermediate in the conversion of CCC to choline (1276).

On foliar surfaces, CCC was absorbed slowly and resided as such. Rats fed CCC excreted the unchanged compound (125, 216).

After application of CCC to wheat and barley, CCC was transformed into a quaternary base which was transported to the roots. In the leaves of winter wheat and spring barley, CCC was metabolized to an unidentified choline derivative (1315).

2,4-D and Related Compounds

2,4-D	2,4,5-T	4-(3-CPB)
2,4-DB	2,4,5-TP	4-(4-CPB)
4-(2,4-DB)	2-CPA	3-(2,4,5-TB)
MCPA	4-CPA	4-(2,4,5-TB)
MCPB	4-(2-CPB)	Phenoxyacetic Acid
		2,4,6-T

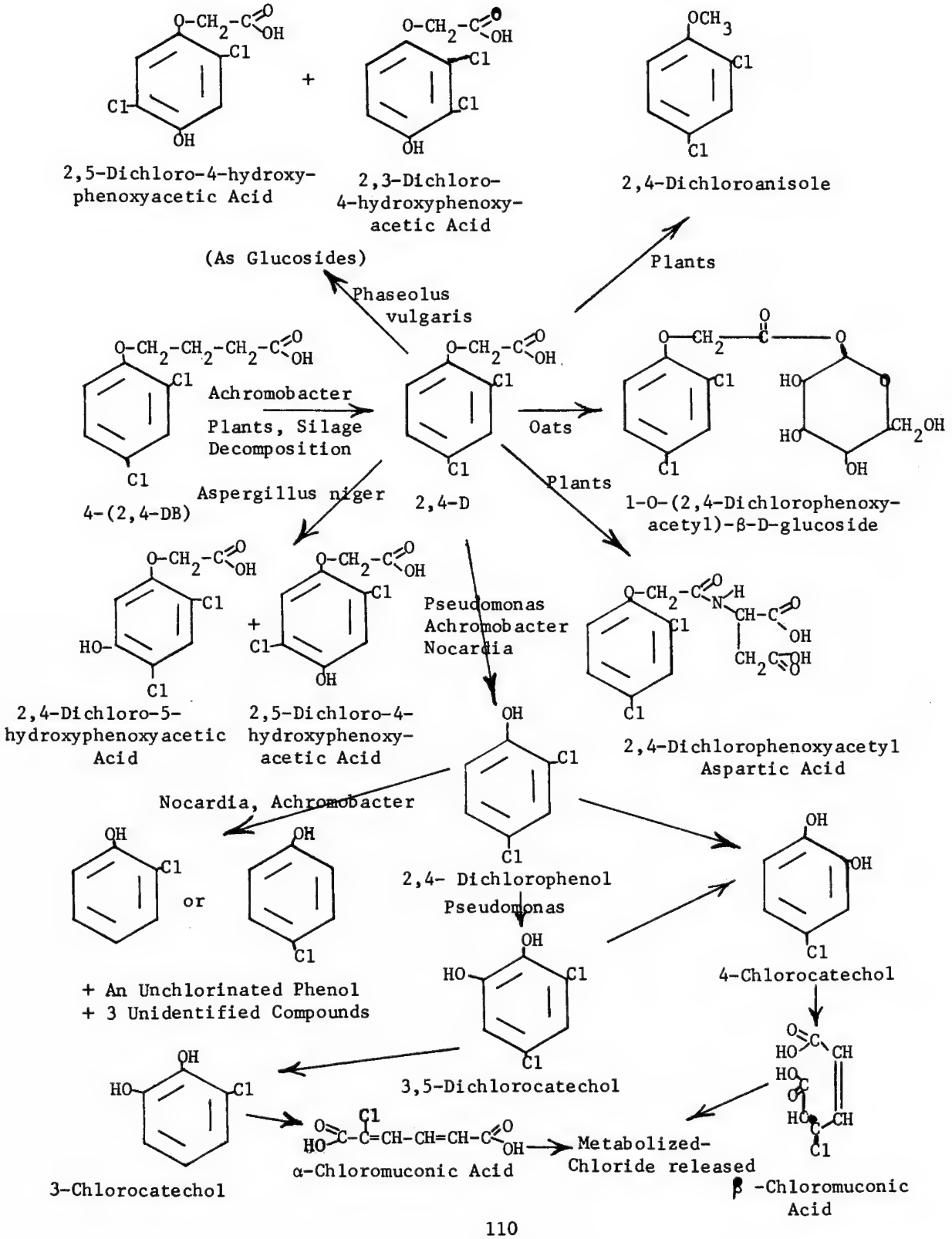
2,4-D[2,4-Dichlorophenoxyacetic acid]

In feeding studies of 2,4-D with dairy cows and steers, (65, 67, 582, 583, 887) 2,4-D was found unchanged in the urine only. No evidence of beta-oxidation was found. Similar findings were obtained with sheep. Ninety-six percent of an orally administered dose of 2,4-D-C^{14} to a sheep was excreted unchanged in the urine in 72 hours and slightly less than 1.4% in the feces. Very little residual radioactivity was found in edible tissue (281).

In rats receiving 1 to 10 mg of 2,4-D, there was almost complete excretion of the herbicide in the urine and feces in 48 hours. At higher dosage levels, some accumulation in tissues occurred. Analyses also indicated that traces of an unidentified metabolite appeared in the urine (785).

After exposure of bean plants (Phaseolus), sun flowers (Helianthus annus), maize (Zea mays) or barley (Hordeum) to 2,4-D, 2,4-dichlorophenol was observed (1559).

Hydrolysis of esters (311, 1040) and decarboxylation (93, 396, 1181) of 2,4-D by plants has also been shown. The free acid has been demonstrated on bean plants, corn plants and forage after treatment with 2,4-D butoxyethanol, propylene glycol butyl, butyl and 2-ethylhexyl esters (423, 431, 600, 634, 802). Treatment of lemons with C^{14} labeled 2,4-D isopropyl-ester indicated that the ester was hydrolyzed and that part of the 2,4-D then reacted with some plant constituent to form an ester-like complex. Ester-like residues were also found after treatment with the sodium, diethanolamine, or triethanolamine salts (413, 414). Samples of fresh citrus peel were prepared by compositing peel samples obtained from oranges from trees sprayed with 2,4-D isopropyl ester. In addition to free acid and ester, a conjugate was also found. The latter became available for extraction only after heat treatment. Preliminary investigations indicated that 2,4-D was conjugated with pectin (966).


2,5-Dichloro-4-hydroxyphenoxyacetic Acid
2,3-Dichloro-4-hydroxyphenoxyacetic Acid
2,4-Dichloroanisole
(As Glucosides)
Phaseolus vulgaris
Plants
Achromobacter
Plants, Silage Decomposition
Oats
4-(2,4-DB)
Aspergillus niger
2,4-D
Plants
1-O-(2,4-Dichlorophenoxyacetyl)-β-D-glucoside
2,4-Dichloro-5-hydroxyphenoxyacetic Acid
2,5-Dichloro-4-hydroxyphenoxyacetic Acid
Pseudomonas
Achromobacter
Nocardia
2,4-Dichlorophenoxyacetyl Aspartic Acid
Nocardia, Achromobacter
2,4- Dichlorophenol
or
Pseudomonas
+ An Unchlorinated Phenol
+ 3 Unidentified Compounds
4-Chlorocatechol
3,5-Dichlorocatechol
3-Chlorocatechol
α-Chloromuconic Acid
Metabolized-Chloride released
β -Chloromuconic Acid

On cotton, cucumbers, beans, and grain sorghum, labeled 2,4-D gave rise to $C^{14}O_2$ (681, 1478, 1481). Pea and tomato plants have also been studied (422). In young leaves and bolls of cotton, material chromatographically different from 2,4,-D was formed. Sorghum converted 2,4-D to a complex different than that found in cotton (1038, 1081, 1322, 1476, 1477, 1478, 1479, 1480, 1481).

Amino acids have been implicated in the formation of some compounds, as in the case of 2,4-dichlorophenoxyacetylaspartic acid (27, 62). Evidence indicated that 2,4-D moved through plants as a protein complex, which could be recovered after aqueous extraction and NaOH hydrolysis, into the roots where most of the degradation occurred (225). Resistant plants were grown in water cultures treated with 2,4-D. Leaves were homogenized and a protein fraction was obtained that contained 2,4-D in a bound form not further identified (273). In big leaf maple (Acer Macrophyllum Pursh), 2,4-D was converted into two metabolites. One of these was the same compound characterized previously (214) as a 2,4-D protein complex which yielded 2,4-D and 12 amino acids on acid hydrolysis (1087).

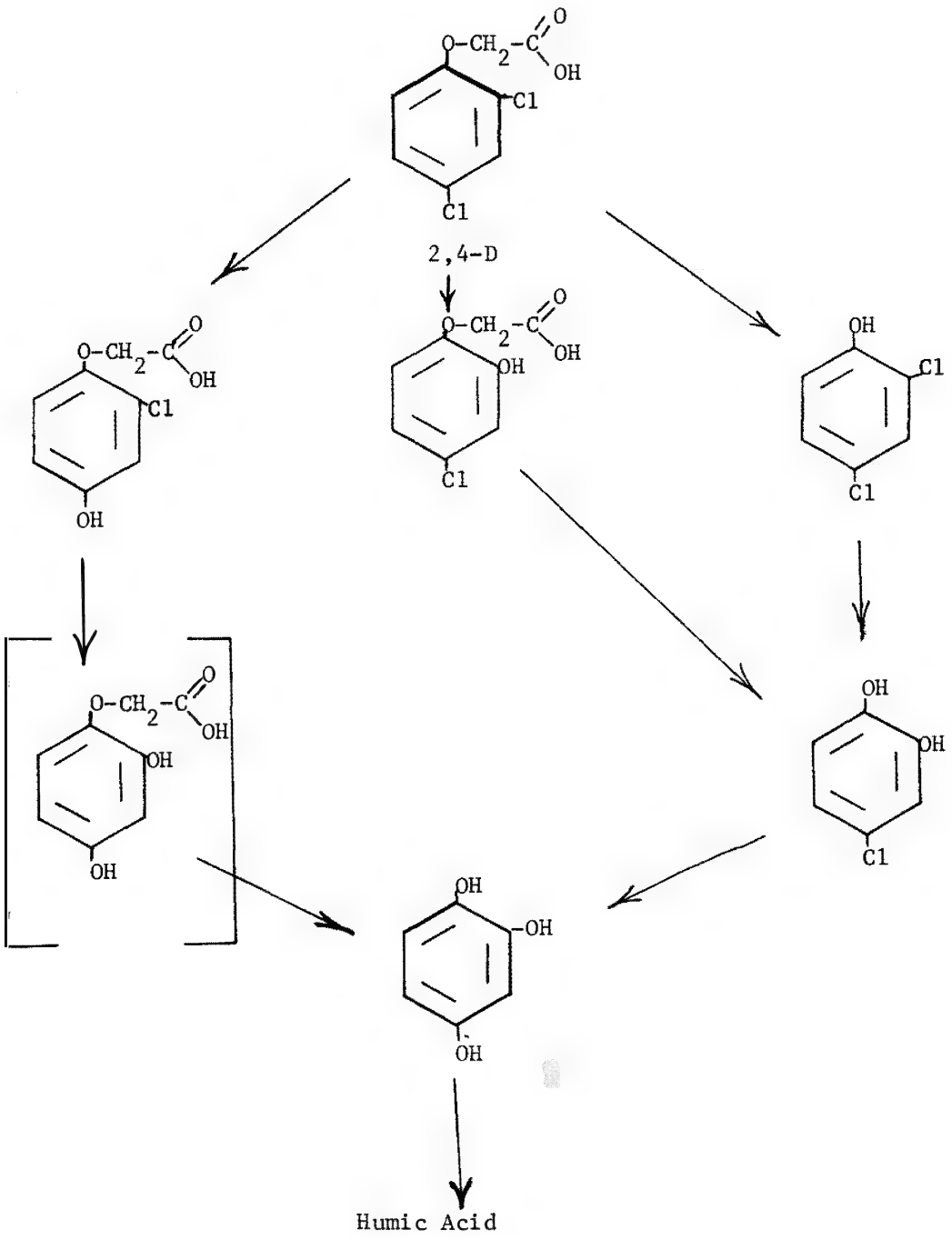
Glucose esters were suggested (314, 798, 799) and studies have shown that glucoside complexes were formed. From stem tissues of oats (Avena sativa), 1-O-(2,4-dichlorophenoxyacetyl)-β-D-glucose was isolated (1401), and from stems of the kidney bean (Phaseolus vulgaris), the 2,5- and 2,3-dichlorophenoxyacetic acid glucosides have been obtained (1400).

From comparative studies with sensitive and insensitive plants, two metabolic paths were proposed involving initial glucose ester formation and oxidative ring cleavage of the aromatic ring to yield monochloroacetic acid (1436). The latter has been detected in plants prior to the onset of treatment symptoms; and it has been suggested that the effect of 2,4-D resulted from the action of monochloroacetate arising from 2,4-D degradation (1436, 1609).

Plants are capable of hydroxylating phenoxyacetic acids (1399, 1498). When bean plants were treated with 2,4-D, three compounds were found (314). One corresponded roughly to that of 2,4-dichloranisole; one was water-soluble, ether-insoluble ester derivative; and the third, an ether-soluble compound with a basic structural change. The methyl derivative was less volatile than 2,4-D methyl ester, but more volatile than the 5-hydroxy-2,4-D methyl ester. It might be one of the other two hydroxy derivatives; however, 6-hydroxy-2,4-D was not detected (60, 420, 680, 726, 727).

The biotransformation of 2,4-dichlorophenoxyalkanoic acids and related compounds by soil microflora has been extensively studied (44, 45, 46, 48 51, 101, 216, 217, 218, 284, 419, 420, 438, 442, 644, 734, 1083, 1349, 1467, 1468, 1469). Phenoxyalkanoic acids with an even number of carbons in the

Photo Decomposition

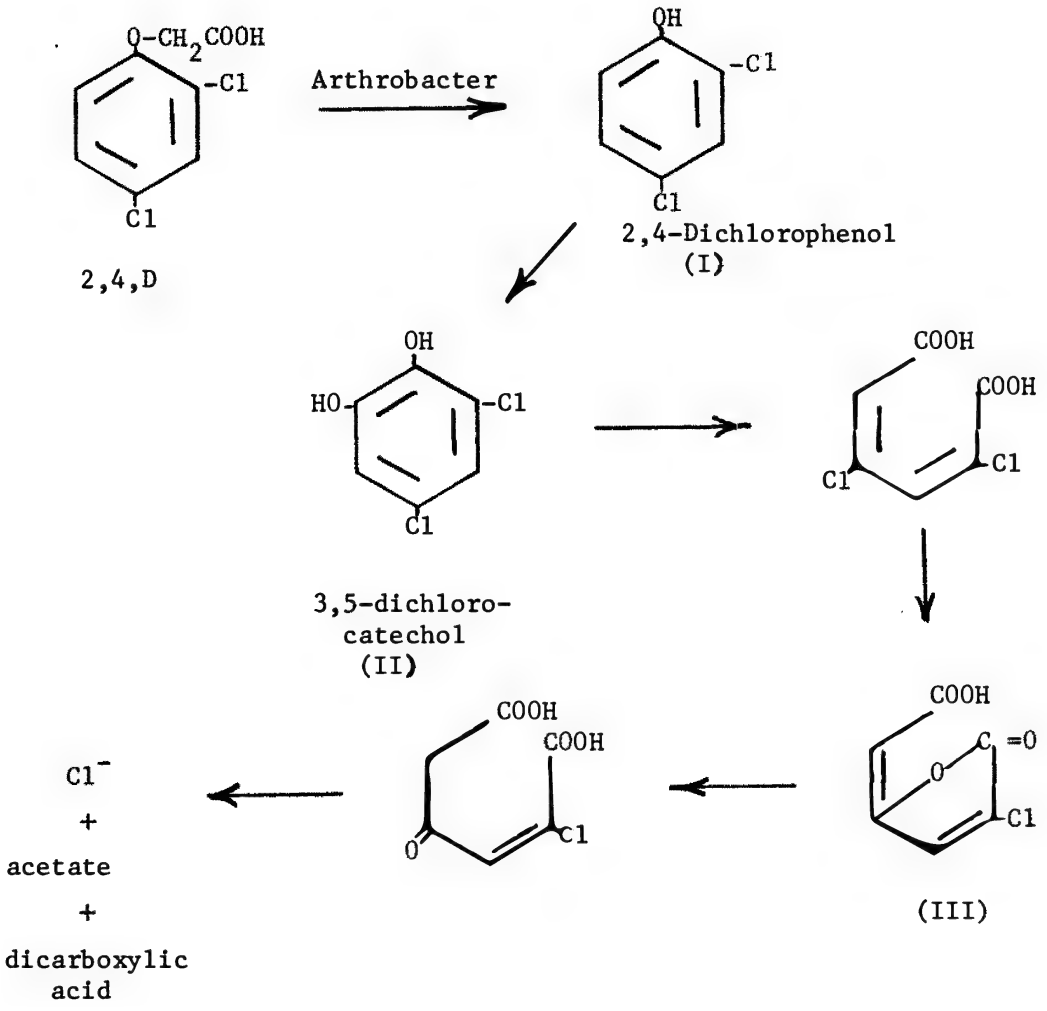


fatty acid were converted by β-oxidation to products with an even number of carbons (580, 588, 864, 1467, 1468, 1469). A second mechanism involved cleavage of the ether linkage (50, 131, 225, 915, 916, 918).

Evidence has been obtained that 2,4-D is dissimilated by a variety of microorganisms (16, 1205) through 2,4-dichlorophenol and 4-chlorocatechol (48). A product from the degradation of 2,4-D by bacteria of the genus Pseudomonas has been identified as β-chloromuconic acid. A second species of Pseudomonas gave rise to α-chloromuconic acid (449). In other studies, 6-hydroxy-2,4-D was reported (894). Pure cultures of a Nocardia species and an Achromobacter strain of bacteria rapidly degraded 2,4-D and the presence of 2,4-dichlorophenol, chlorohydroquinone, a monochlorophenol, an unchlorinated phenol and three other unidentified compounds have been demonstrated (46, 101, 102, 438, 1083, 1350, 1351, 1392). The main product of 2,4-D metabolism by the mold Aspergillus niger van Tiegh was 2,4-dichloro-5-hydroxyphenoxyacetic acid. By means of infrared and mixed melting points, a second metabolite was identified as the 2,5-dichloro-4-hydroxyphenoxy-acetic acid--the first time such a rearrangement was reported. Another unidentified acid, not the 3- or 6- hydroxyacid, was also found (439, 440). Arthrobacter sp. degraded 2,4-D via 2,4-dichlorophenol and 2,4-dichloroanisole (895, 896). In excess of 80% of the chloride was released in a 3 hour incubation period with crude extracts or the soluble fraction (894). A Corynebacterium species also degraded 2,4-D with quantitative release of chloride (1235). In natural surface waters, 2,4-D isopropyl and butyl esters were hydrolyzed to 2,4-D and their respective alcohols (18). When triethanolamine salts of C^{14}-carboxy labeled 2,4-D were applied in water to forest litter, liberation of $C^{14}O_2$ was rapid (1086).

In the presence of water and ultraviolet light, 2,4-D decomposed rapidly with formation of 2,4-dichlorophenol. This underwent further decomposition to 4-chlorocatechol, polymeric humic acids and chloride. Some 2-hydroxy-4-chlorophenoxyacetic acid and a very small amount of 2-chloro-4-hydroxy phenoxyacetic acid were present (317, 611, 1434, 1435). In the presence of riboflavin, compounds containing more than one aromatic nucleus were probably also formed in addition to 2,4-dichlorophenol. Products differed according to the original pH and concentration of the treated solution (100, 611).

In studies with a soil arthrobacter sp., extracts catalyzed the removal of the side-chain to form 2,4-dichlorophenol (I). Enzyme systems present were able to metabolize the phenol and the accumulation of 3,5-dichlorocatechol (II) was demonstrated. The degradation product of the catechol was identified by mass spectra and infra-red as α-chloro-γ-carboxymethylene-Δ$^{\alpha}$-butenolide (III) (1679, 1691, 1692).

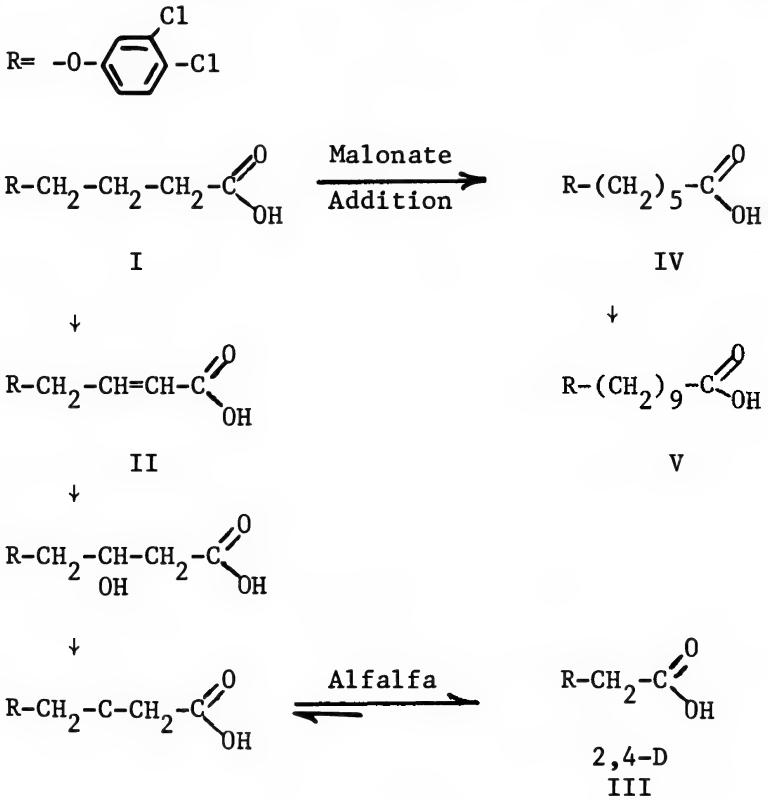


4-(2,4-DB) [4-(2,4-Dichlorophenoxy) butyric acid]

In laboratory tests, bluegill (Lepomis gibbosus) converted 4-(2,4-DB) to 2,4-D (585). When 4-(2,4-DB) was fed to dairy cows, however, 2,4-D was not found, although it does not decompose in the rumen. The major portion of 4-(2,4-DB), therefore, was presumably degraded by a mechanism other than β-oxidation in the cow (584, 887). In silage (881, 882) timothy, birdsfoot trefoil, or pea plants (450), decomposition of 4-(2,4-DB) has been shown to proceed by β-oxidation. In Bigleaf maple (Acer macrophyllum Pursh), the primary oxidation product of 4-(2,4-DB) was 2,4-D (1088).

Studies with 4-(2,4-DB) also showed that it was detoxified by Flavobacterium sp. (917), releasing chlorine and cleaving the ring (206). Aspergillus niger metabolized 4-(2,4-DB) to 2,4-D and four unidentified phenolic acids (441).

After treatment of alfalfa (Medicago sativa L.) with 2,4-DB, extracts were analyzed. Metabolites of 2,4-DB (I) as determined by gas chromatography included the corresponding dichlorophenoxy-caproic (IV) and decanoic (V) acids as well as small amounts of the crotonic (II) and acetic (III) acid analogs. Indications of analogs with longer side chains than decanoic were also observed (1693).

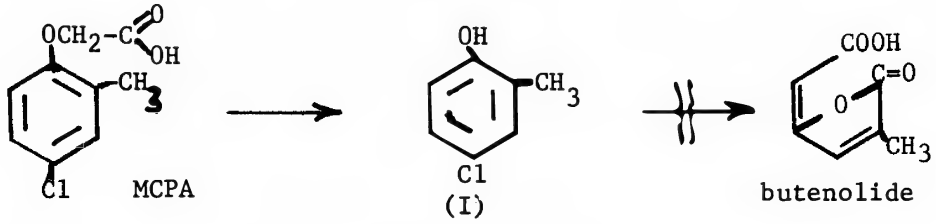


MCPA [4-Chloro-2-methylphenoxyacetic acid]

Arthrobacter sp. and flavobacterium peregrinum degraded MCPA to 4-Chloro-2-methylphenol. The flavobacterium further oxidized the phenol to release all chloride (48, 133, 894). Aspergillus niger van Tiegh also metabolized MCPA to 4-chloro-2-methyl-5-hydroxyphenoxyacetic acid (439, 440).

In other studies, a gram-negative soil bacterium metabolized MCPA to 5-chloro-o-cresol, a compound believed to be 6-hydroxy-MCPA and α-methyl-γ-carboxymethylene-Δ^{α}-butenolide. A pathway for microbial degradation was suggested (529).

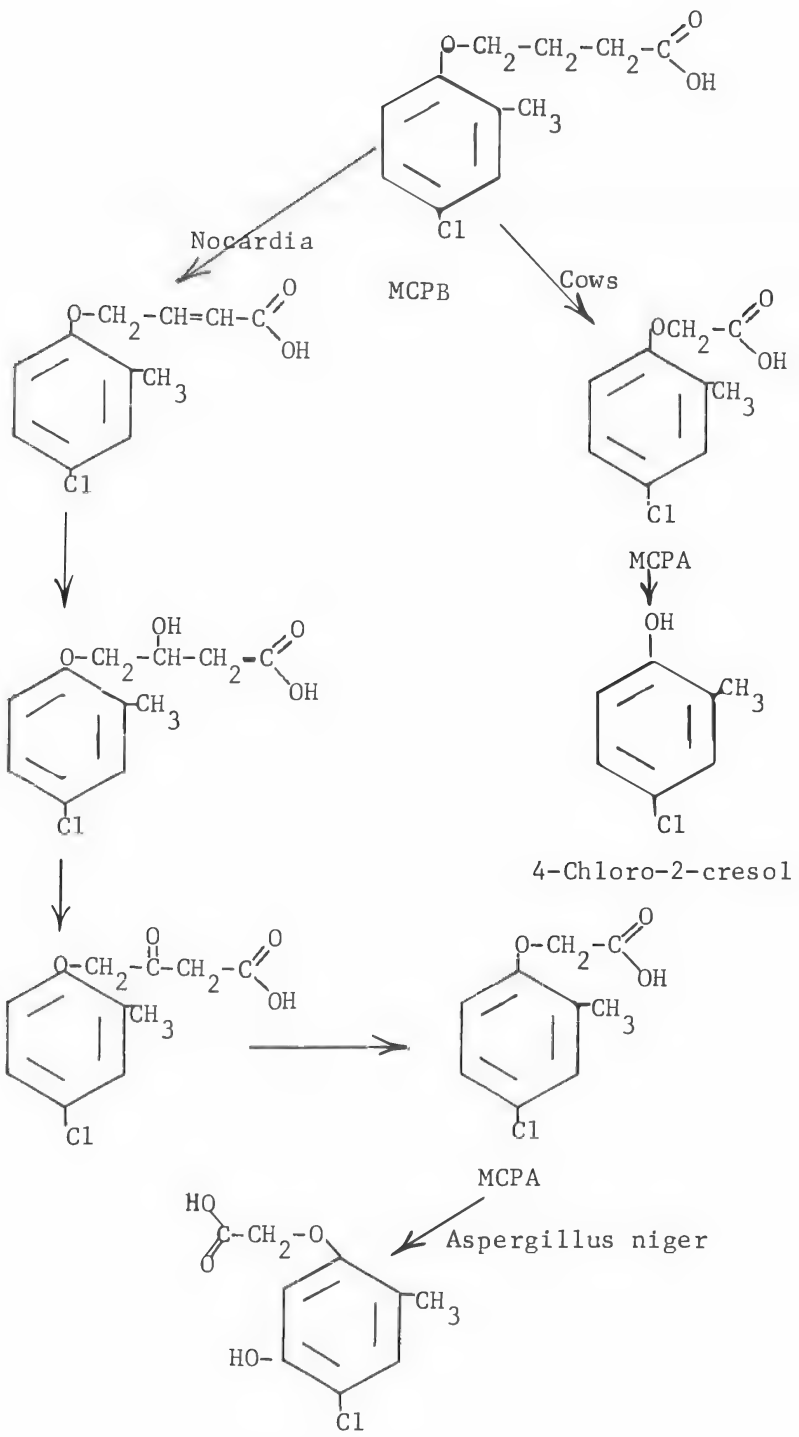
Ultraviolet (3407 to 3888 Angstroms) activated MCPA (1137). Enzymes from an arthrobacter species catalyzed the removal of the side chain with formation of 2-methyl-4-chlorophenol [5-chloro-o-cresol] (I) (1679).



MCPB [4-(4-Chloro-2-methylphenoxy) butyric acid]

Degradation of compounds related to 2,4-D has also been studied (1460). MCPB gave rise to MCPA when fed to dairy cows (65, 66, 67).

Aspergillus niger and Nocardia opaca degraded MCPB to MCPA and 4-(4-chloro-2-methylphenoxy)-3-hydroxybutyric acid (441, 1468, 1469).



2-Methyl-4-chloro-
5-hydroxyphenoxyacetic Acid

2,4,5-T [2,4,5-Trichlorophenoxyacetic Acid]

Cows fed 2,4,5-T excreted it as a soluble salt in their urine (1259).

When Winesap and Staymen Winesap cultivars were exposed to 2,4,5-T, some decarboxylation occurred (1181). Bean plants (Phaseolus), sun flowers (Helianthus annus) and barley (Hordeum) converted 2,4,5-T to its phenol (1559). 2,4,5-T was also decarboxylated by woody plants (93).

From comparative studies with sensitive and insensitive plants, two paths were proposed, involving initial glucose ester formation and oxidative ring cleavage to yield monochloroacetate. The latter was detected in treated plants prior to the onset of treatment symptoms (1436).

Sweetgum (Liquidambar styraciflua L.) and southern red oak (Quercus falcata Michx.) were sprayed with an aqueous homogenate of 2,4,5-T n-butyl ester. After one month, leaves were collected and assayed using gas chromatography to detect residues. 2,4,5-Trichlorophenol was observed but no evidence was found to indicate formation of 2,4,5-Trichloranisol (460, 461). After application to Bigleaf maple (Acer Macrophyllum Pursh) and mesquite seedlings, 2,4,5-T was metabolized but the products were not identified (1043, 1087).

Triethanolamine salts of C^{14} -carboxy labeled 2,4,5-T were applied in water to the surface of some collected forest litter. Liberation of $C^{14}O_2$ was slow but increased with time (1086).

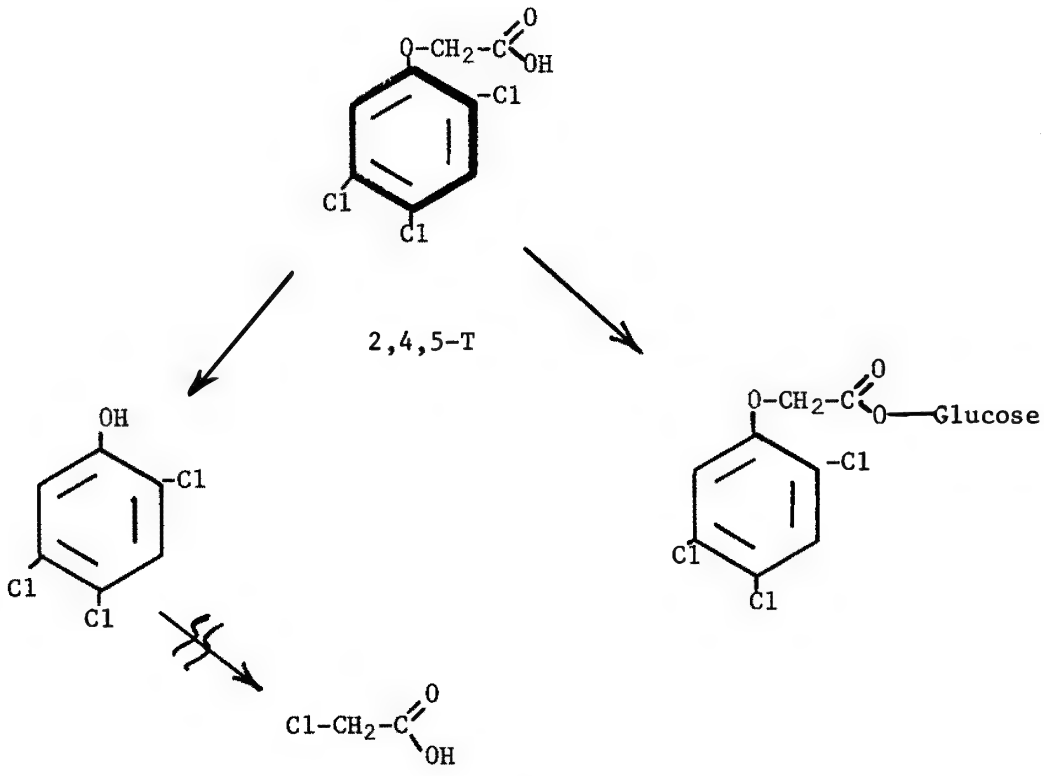
2,4,5-TP (Silvex) [2-(2,4,5-Trichlorophenoxy)propionic Acid]

When fed to cows, silvex was excreted as a soluble salt in the urine. Kuron, the propylene glycol butyl ether ester of silvex, was hydrolyzed prior to elimination (1259).

Samples of fresh citrus peel were prepared by compositing peel samples obtained from oranges from trees sprayed with 2,4,5-TP propylene glycol butyl ether ester. In addition to free acid and ester, a conjugate was found. The latter became available for extraction only after heat treatment. Preliminary investigations indicated that 2,4,5-TP was conjugated with pectin (966). Decarboxylation of 2,4,5-TP by prickly

pear (Opuntia spp.) was 1/2 to 1/3 of that by soybean. In addition to unaltered 2,4,5-TP, at least four labeled metabolites were observed after application of silvex-1-C^{14} was applied to prickly pear (277, 278).

When the propylene glycol butyl ether ester was applied to water overlying various soil types, the herbicide was hydrolyzed almost totally to the acid in about two weeks. Adsorption of the acid by the soils was also indicated (1725).

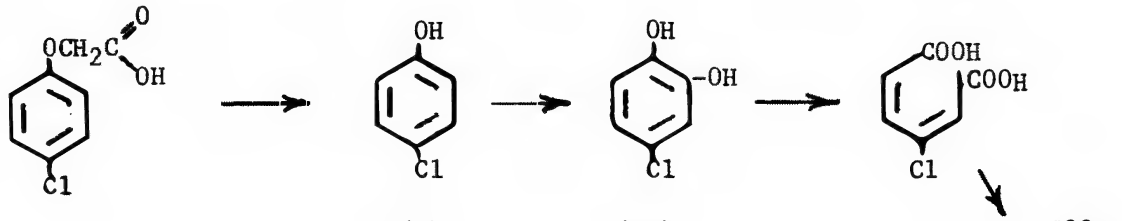


monochloroacetate

2- and 4-Chlorophenoxyacetic Acid (2-CPA and 4-CPA)

Both compounds were degraded by arthrobacter sp. to their respective phenols. Some chloride was also released (894).

4-Chlorophenol (I) was metabolized to 4-chlorocatechol (II) and α-carboxymethylene-Δ^{α}-butenolide (III) (1679, 1691, 1692).



(I) (II) (III)

4-(2-CPB) [4-(2-Chlorophenoxy) butyric Acid] (I)

4-(3-CPB) [4-(3-Chlorophenoxy) butyric Acid] (II)

4-(4-CPB) [4-(4-Chlorophenoxy) butyric Acid] (III)

Using aspergillus niger:

Metabolism of I gave rise to 4-(2-chlorophenoxy)-3-hydroxybutyric acid, 4-(2-chloro-4-hydroxyphenoxy) butyric acid, 2-chlorophenoxyacetic acid, 2-chloro-4-hydroxyphenoxyacetic acid and 2-chloro-3-hydroxyphenoxyacetic acid (441).

Metabolism of II produced 4-(3-chlorophenoxy)-3-hydroxybutyric acid, 3-chlorophenoxyacetic acid and 3-chloro-6-hydroxyphenoxyacetic acid (441).

Metabolism of III produced 4-(4-chlorophenoxybutyric acid, 4-chlorophenoxyacetic acid, 4-chloro-2-hydroxyphenoxyacetic acid, 4-chloro-3-hydroxyphenoxyacetic acid (441).

3-(2,4,5-Trichlorophenoxy) butyric acid [3-(2,4,5-TB)]

3-(2,4,5-Trichlorophenoxy)butyric acid was metabolized to its acetic acid derivative in wheat but not in pea stems (75).

4-(2,4,5-Trichlorophenoxy)butyric acid [4-(2,4,5-TB)]

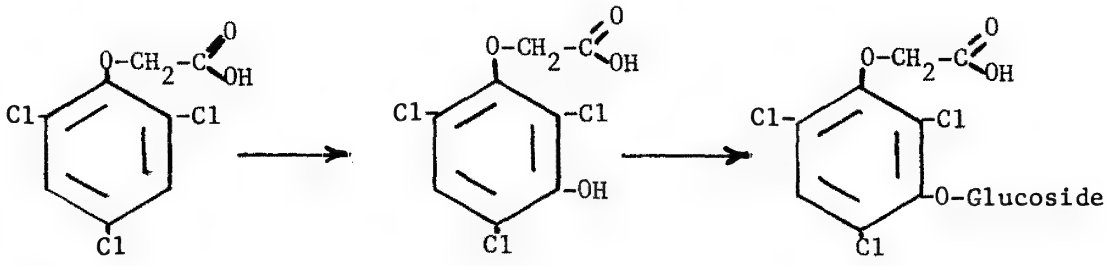
This material was metabolized by aspergillus niger primarily to 3-hydroxy-4-(2,4,5-trichlorophenoxy)butyric acid, which, when used as a substrate, gave only a trace of 2,4,5-trichlorophenoxyacetic acid (441).

Phenoxyacetic Acid

Phenoxyacetic acid was hydroxylated mainly in the "ortho" position by aspergillus niger van Tiegh (131).

2,4,6-T [2,4,6-Trichlorophenoxyacetic Acid]

After exposure of the oat Avena sativa to 2,4,6-T, hydroxylation occurred at the 3-position and a glucoside was isolated and identified (1401).

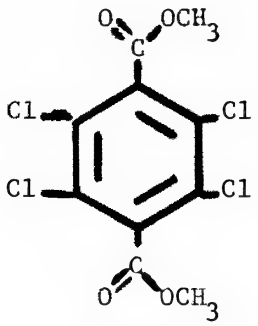


2,4,6-Trichloro-
phenoxyacetic Acid

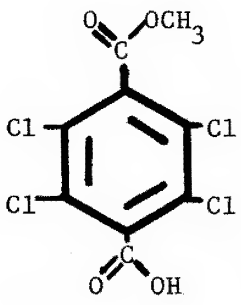
Dacthal [2,3,5,6-Tetrachlorodimethylphthalate]

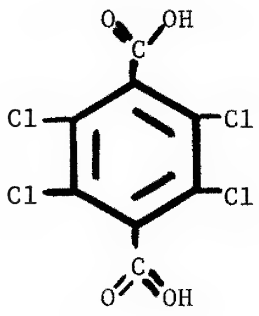
No residues of dacthal were found in milk of dairy cows fed this compound. About one per cent of intact dacthal was found in the manure. Dacthal monoacid, 2,3,5,6-tetrachloromonomethylterephthalate, was eliminated via manure (3.3%) and urine (90.9%). Beef liver slices rapidly produced this same metabolite. Incubation of dacthal with rumen contents did not decompose dacthal (586, 589).

After application of dacthal to soil, the monomethyl and free dicarboxyl analogs were tentatively identified by gas chromatography (537, 1314). Under conditions of gas chromatography, the monomethyl derivative underwent decarboxylation (1314).



Dacthal





Dalapon (2,2-Dichloropropionic acid)

When dairy cows were fed dalapon, less than 1% of the ingested dose appeared as residues in the milk, mainly as unchanged dalapon but with traces of dalapon glycerides (848).

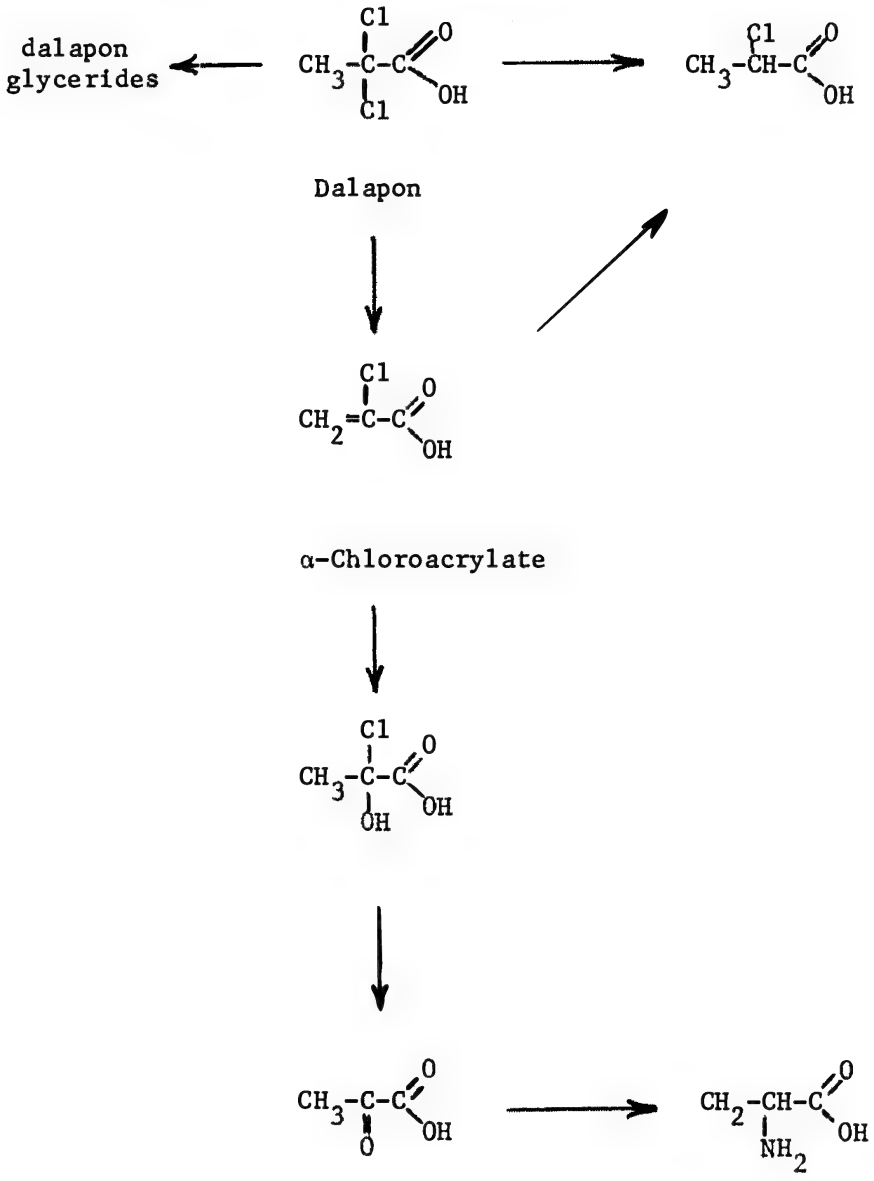
Tracer studies with dalapon-2-C^{14} and Cl^{36} showed that dalapon was absorbed, translocated, and accumulated in higher plants as the original molecule or dissociable salt and might remain non-metabolized for long periods (22, 124, 469, 470, 471, 472, 473, 474, 856). A small amount of decomposition occurred slowly and resulted in release of Cl^{36} or incorportation of C^{14} into other compounds. Tests with tissue homogenates did not indicate metabolic incorporation of dalapon into insoluble macromolecules. Some indication of dehalogenation was indicated on chromatograms (470, 473). Propionic acid, which could arise through such reactions, competes with β-alanine in pantothenic acid synthesis. Studies have shown that dalapon does, in fact, inhibit pantothenate synthesis (661).

Many microorganisms have been found capable of dissimilating dalapon (355, 666, 667, 668, 685, 731, 914, 920, 1205) with release of chloride and formation of monochloropropionate (470, 472, 473, 732).

Pseudomonas dehalogens, nocardia sp., agrobacterium sp. and arthrobacter sp. released chloride (665, 668, 732). The latter organism also gave rise to pyruvate (776, 777) and incorporated 2-carbon units into cellular components (96). The 1-C^{14} label was found in protein and lipid. When 2-C^{14} label was used, the label appeared in the lipid, protein and nucleic acid fractions. Pyruvate and alanine were shown to be the early metabolites (96).

Studies with cell free systems indicated metabolism of dalapon to α-chloroacrylate, α-chloro-α-hydroxy-propionate and finally pyruvate (776, 777).

Anything affecting soil microbial populations may affect persistence of organic herbicides. Organic matter, pH, and cation exchange capacity are important factors affecting microbial decomposition of dalapon (685, 764).

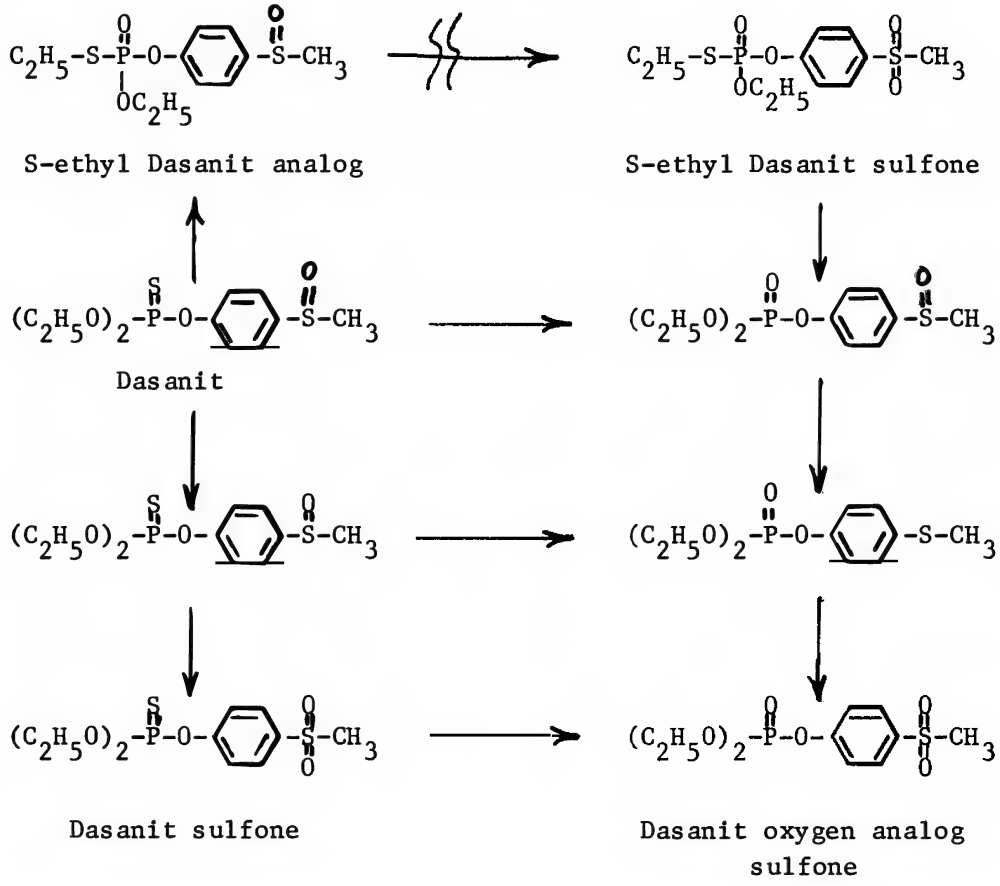

dalapon glycerides
Dalapon
α-Chloroacrylate
Pyruvic acid
Alanine

Dasanit (Bayer 25141) [0,0-diethyl-0-p(methylsulfinyl)phenyl phosphorothioate]

P^{32}-labeled dasanit was applied to the stems of young cotton plants or to the roots of plants by immersion of roots in water dispersions. In the case of the stem application, more than 80% of the radioactivity was in the form of the parent compound. The major metabolites were the oxygen analog and dasanit sulfone. After nine days, a small amount of the oxygen analog sulfone was also detected (762).

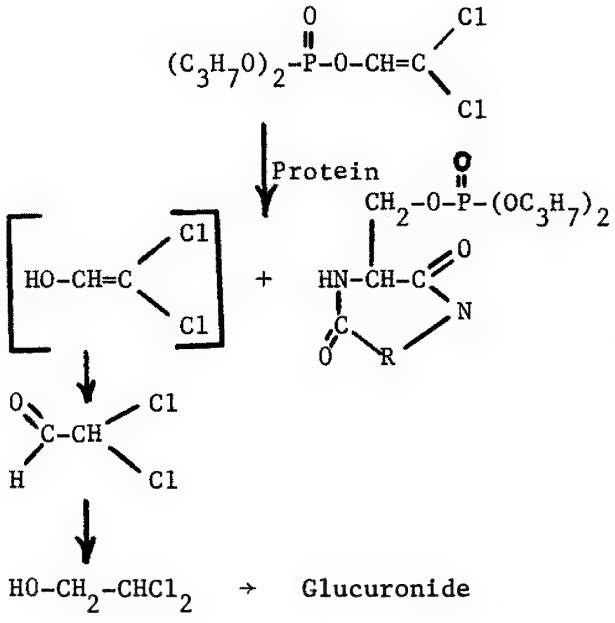
Dasanit underwent slow oxidation in air to form the sulfone 0,0-diethyl 0-p-methylsulfonylphenyl phosphorothionate. In addition to this, the characteristic isomerization of phosphorothionates to phosphorothiolates also occurred.

Metabolism studies with roaches have indicated the presence of the isomeride and sulfone of Dasanit and the S-ethyl isomer of the sulfone (104, 105).



DDP (di-n-propyl-2,2-dichlorvinyl phosphate)

Enzymatic studies with chymotrypsin have shown that DDP phosphorylated protein without releasing acid. At the same time, dichloroacetaldehyde was liberated (718). Studies with DDVP have shown that the aldehyde may then be metabolized to the saturated alcohol and conjugated as the glucuronide.. (See DDVP).



DDT [1,1,1-Trichloro-2,2-bis(p-chlorophenyl)ethane]
DDD (TDE, Rhothane) [1,1-Dichloro-2,2-bis(chlorophenyl)ethane]
Dicofol [1,1-bis(p-chlorophenyl)-2,2,2-trichloroethanol] (Kelthane)

From mammals fed to exposed to DDT (236, 319, 636, 945, 1242, 1342, 1367, 1494, 1662) DDE, DDA and dichlorobenzophenone have been isolated and identified. Using DDT-C^{14}, DDE has been found in both bile and feces (207, 735). DDA, both complexed and free, has been found. Stability to alkaline hydrolysis and negative qualitative tests indicated that the complex was not a glucuronide but did suggest an amide linkage such as in hippuric acid.

Studies with beef cattle suggested that DDT was dechlorinated to DDD in the rumen. Beef steers stored DDT, DDD and DDE when DDT was administered orally. The ration of stored DDD and DDT was much lower in steers that received DDT intraperitoneally and intravenously (950). Perfused bovine livers converted DDT to DDD and DDE (1705).

A pathway for the metabolism of DDT in rats (337, 455, 801, 1156) and yeast (753) has been proposed on the basis of studies with DDT and related compounds. These studies indicated DDD as an intermediate. A phenolic compound was also implicated (1036). Studies suggested that the major fecal products in rats consisted of DDA conjugated with cholanic acid or amino acids (735). After administration of p,p'-DDT in oil and p,p'-DDA in water to rats via stomach tube, a conjugate of p,p'-DDA was isolated from feces and urine. This conjugate contained one molecule each of serine and aspartic acid together with 8 molecules of water of hydration per molecule p,p'-DDA. The presence of four other metabolites was also indicated, possibly identical with (1) p,p'-dichlorobenzhydrol; (2) p,p'-dichlorodiphenylmethane; (2) p,p'-dichlorobenzophenone; (4) DDE (1161, 1162). DDA was found in tissues of rats and rabbits after intraperitoneal, oral and intravenous administration of DDT (747). Other studies have shown that rat livers degrade DDT to DDD and DDE (1445, 1662).

When fed DDT, monkeys did not store easily detectable DDE, in contrast to the situation in man and the rat. However, when DDE was fed, the monkey accumulated DDE. Since all three species excreted some DDA, it must be assumed that DDA can be produced without DDE as an intermediate metabolite (389). Recent studies have also shown that, when o,p'-DDT was fed to rats, a biological isomeric transformation of o,p'-DDT to p,p'-DDT occurred (800, 801).

The metabolism of DDT by the common grackle, Quiscalus quiscala, showed that orally ingested DDT was slowly absorbed from the gut and rapidly metabolized. Unabsorbed DDT was eliminated rapidly. In addition to DDT, both DDD and DDE were found in tissue extracts and the feces. In vitro studies, using liver cultures free from bacterial contamination, also showed metabolism of DDT to DDD and DDE (1463, 1464).

The proportion of p,p'-DDT to p,p'-DDE in livers of sacrificed Bengalese finches was related to the length of storage in the refrigerator. This conversion of DDT to DDE occurred under anaerobic conditions at 37°C and at -11.5 to 14.5°C (728, 729). It was thought that this degradation was caused by bacteria. However, recent studies with avian frozen blood tissues indicated non-enzymatic degradation (394). DDD was formed anaerobically whereas DDE formed under aerobic conditions (204).

In pigeons (Columba liva), feeding of DDT gave rise to DDE and DDD by separate pathways. Whereas feeding of DDE did not give rise to DDD, feeding of DDD did give rise to small residues of DDE. The DDD was rapidly metabolized exclusively to DDMU. Pigeons fed DDMU showed residues of this material and these residues declined rapidly. Gas-liquid chromatography revealed the presence of an unknown compound. After isolation by preparative scale gas-liquid chromatography, infra-red and mass spectroscopy showed the compound to be identical with 1,1-bis(p-chlorophenyl)ethane. This material could arise metabolically from DDMU or after prolonged exposure to light during and after extraction. Transformation by gut flora has been demonstrated and could also give rise to this ethane analog (1749, 1763).

DDT, DDD and DDE (all p,p'-)in peanut oil were separately injected into fertile leghorn eggs or fed in the diet to chicks hatched from untreated eggs. No significant differences in the pattern of metabolites between the two treatments were observed. DDT gave rise to o,p'-DDT, DDE, DDD (p,p'- and o,p'-), DDMU, DDMS, DDNU, DDOH, DDA, DDM, and DBP. p,p'-DDD gave rise to o,p'-DDD, DDMU, DDMS, DDNU, DDOH, DDA, DDM, and DBP. DBP was detected as a metabolite of DDE (1738).

Studies with rainbow trout, Salmo gairdneri, showed that the liver converted DDT to DDE (1473). From bullhead fish, 1,1'-bis-p-chlorophenyl-2-chloroethylene was obtained (180). Most metabolism in fish resulted from the action of intestinal microflora which converted DDT to DDE and/or DDD (1473, 1647). In vitro studies with carp blood and DDT showed some conversion to DDD, DDE and possibly DDMU (1657). Atlantic salmon, Salmo salar, degraded DDT to DDE and DDD (1726, 1768, 1770); Salmo clarki, also(1769).

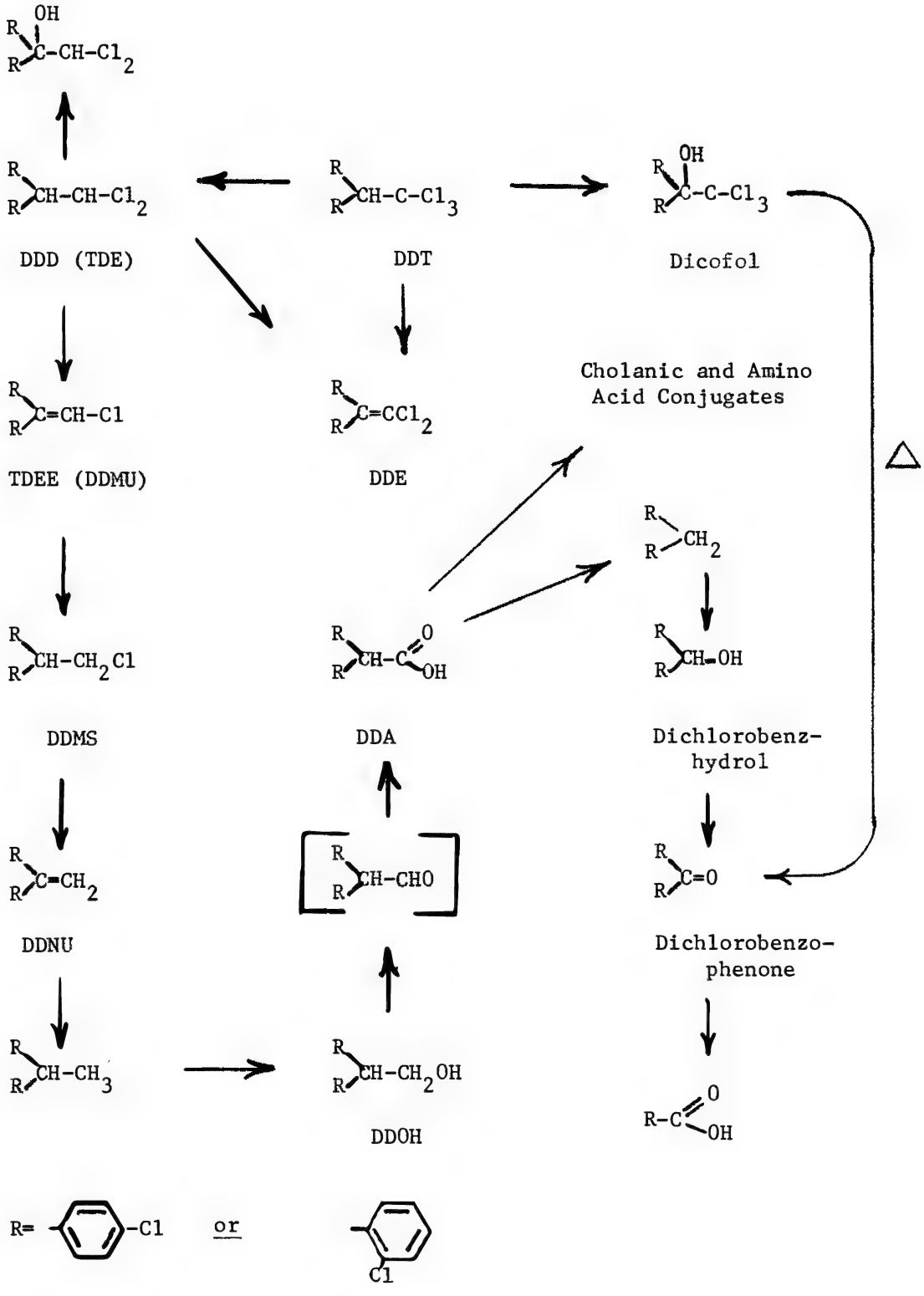
Detoxification has long been considered a major factor in the defense of insects against the lethal action of DDT and this concept was strengthened by the isolation of DDT-dehydrochlorinase, a glutathione dependent enzyme (885, 886, 1014, 1359). In many insects this system catalyzed the degradation of p,p'-DDT, to p,p'-DDE or the degradation of p,p'-DDD to its corresponding ethylene derivative (4, 58, 59, 198, 463, 679, 880, 884, 1101, 1148, 1149, 1150, 1151, 1152, 1154, 1354, 1355, 1356, 1358, 1360, 1386, 1614, 1655). o,p'-DDT was not degraded.

Investigations have shown that DDT may be metabolized by insects to as many as 7 metabolites (unidentified) in addition to DDE, most of which are more polar than DDT (695, 878).

The housefly, Musca domestica L., converted DDT to DDE and unidentified water soluble products possibly conjugated with a carbohydrate (1147, 1265, 1396, 1518). Fly homogenates, in the presence of $NADPH_2$, converted DDT to kelthane (1427).

American, Periplaneta americana L., and German, Blatella germanica L., cockroaches oxidized DDT to DDE and other water soluble products which, upon acid hydrolysis, could be extracted by ether (288, 693, 1217, 1518). It was thought that the water soluble material was a conjugate composed of a DDT metabolite and a carbohydrate (215, 1448). In the American cockroach, dichlorobenzophenone and what was believed to be dichlorobenzhydrol also were detected in the feces (1518). From the German cockroach, an enzyme system was prepared that oxidized DDT to a compound with the same chromatographic characteristics as dicofol (5, 527). Dichlorobenzophenone was also found in tissues after exposure of German cockroaches to DDD (527). Other studies indicated that various nerve components of both American and German cockroaches formed a complex with DDT (635, 942).

Fifth-instar larvae of Aglais urticae (L.) (Lepidoptera: Nymphalidae) and fourth-instar larvae of Chorthippus brunneus (Thunberg) (Saltatoria: Acrididae) metabolized DDT to DDE (1747).


DDD (TDE)
DDT
Dicofol
Cholanic and Amino Acid Conjugates
TDEE (DDMU)
DDE
DDMS
DDA
Dichlorobenz-hydrol
DDNU
Dichlorobenzo-phenone
DDOH
R= -Cl or Cl

Larvae of Culex tarsalis, Culex pipiens fatigans, Culex pipiens quinquefasciatus and Aedes aegypti metabolized DDT to DDE. Generally, some DDD was also produced. With C. fatigans, Aedes aegypti, and several strains of C.p. quinequefasciatus, the water extracts also contained a product that appeared to be the dehydrochlorinated product of DDD (386, 688, 754, 793, 794, 1612, 1613, 1648). Other studies showed that the DDT-resistant mosquito Culex tarsalis Coquillett degraded TDE to DDA, bis(p-chlorophenyl) methanol (DBH) and p-chlorobenzoic acid (1169).

Some studies indicated that metabolism of DDT might not always proceed through DDE. Using a DDT-resistant strain of the human body louse (1144), it was found that DDT was metabolized to a water-soluble compound that behaved like p-chlorobenzoic acid. In subsequent studies, DDT was metabolized to dichlorobenzophenone (DBP) via DDA or to DDE (1154).

Enzyme fractions have been prepared from extractions of human body louse. These fractions indicated the existence of different native enzymes which produce different metabolites. Fraction A gave a 6:1 ratio of neutral to acidic metabolite. The neutral metabolite was predominantly DDE. Fraction B gave a 1:1 ratio of neutral to acidic metabolite. The neutral metabolite in this case was predominantly 4,4'-dichlorobenzophenone. The acidic metabolite produced by both enzyme fractions appeared to be DDA (1011).

In the fruit fly, Drosophila melanogaster, the hydroxy analog of DDT (dicofol) was formed (982, 1424, 1425, 1426). The lady bettle, Coleomegilla maculata de Geer, metabolized DDT essentially to DDE (41).

The milkweed bug, Oncopeltus fasciatus (Dallas) stored unchanged DDT in its internal tissues and excreted it as such. Some water soluble oxidation products were indicated but not identified (1518). When DDA was applied topically, several unidentified compounds were formed (447).

The Mexican bean beetle, Epilachna varivestis Muls, contained DDT-dehydrochorinase activity in its tissues and was able to dehydrochlorinate DDD (1411). When third-instar larvae of Triatoma infestans were topically treated with DDT-C^{14}, two metabolites were detected, kelthane and a compound that behaved like 4,4'-dichlorobenzohydrol. The latter also appeared after topical treatment with kelthane (6).

In the larvae of the tobacco budworm, Heliothis virescens (F.), the predominant product of DDT metabolism was DDE. However, DDA was also present (1449, 1450). The tobacco hornworm degraded TDE to its hydroxy analog (527). It was also found that topically applied DDA was rapidly converted to several unidentified compounds by the hornworm (447). DDT was metabolized to DDE by the desert locust (1344) and the Asian blood-sucking leech, H. nipponia (795).

Studies have indicated that DDD appears in those samples which are permitted to decompose some time after sacrifice and not in those samples analyzed immediately (80). An isolate, Proteus vulgaris, from a mouse intestinal flora converted DDT to DDD (82). This organism, commonly found in the gut of mice and other animals, quickly invades dead tissue and could be assumed to be an agent, if not the sole one, in the conversion of DDT to DDD in animals exposed to DDT. DDD, in turn, was metabolized by Proteus vulgaris to three compounds: (1) 1,1'-bis-(p-chlorophenyl)-2-chloroethane; (2) 1,1'-bis-(p-chlorophenyl)-ethane; (3) 1,1'-bis-(p-chlorophenyl-2-chloroethylene;(81).

It has been shown that bovine rumen fluid was capable of converting DDT to DDD (1012), and that bacteria from the intestines of flies (Stomoxys calcitrans L.) converted DDT to DDE and DDD (1352).

Aerobacter aerogenes converted p,p'-DDT and o,p'-DDT to the corresponding DDD (974). Studies to elucidate the metabolic pathway involved in degradation of DDT indicated that DDD, DDMU DDMS, DDNU DDOH, DDA and DBP were formed (1470, 1471, 1472). Using 2-deuterioethane-labeled DDT, the direct formation of DDD from DDT was demonstrated (1626, 1680).

Extensive studies showed that many microorganisms were capable of metabolizing DDT to DDD primarily. Some DDE was generally found also (258, 738, 852, 975, 1554):

Achromobacter sp.
Aerobacter aerogenes
Agrobacterium tumefaciens
Bacillus cereus
Bacillus cereus mycoides
Bacillus subtilis
Clostridium pasteurianum
Clostridium perfringens
Corynebacterium michiganense
Escherichi coli
Erwinia amylovora
Erwinia ananas
Erwinia carotovora
Erwinia chrysanthemi
Erwinia sp.
Kurthia zopfii
Pseudomonas aeruginosa
Pseudomonas fluorescens
Pseudomonas glycinea
Pseudomonas marginalis
Pseudomonas mors-prunorum
Pseudomonas syringae
Pseudomonas tabaci
Streptococcus bovis
Streptococcus durans
Streptococcus faecalis
Streptococcus faecium
Streptococcus liquifaciens
Streptococcus zymogenus

Several unidentified organisms of the genera bacteroides, lactobacillus, sarcina, aeromonas and yeast were also capable of metabolizing DDT to DDD and/or DDE. Micrococcus did not degrade DDT (1554, 1647).

Studies were conducted with the mold Fusarium oxysporum (Schl.) to elucidate the metabolic pathway. DDD was formed directly and from DDE. DDMU, DDOH, DDA, and DCB were also observed. Formation of the aldehyde (DDHO) was presumed on the basis of indirect proof. The formation of DDOH and DDA was suggested as arising from dismutation of the aldehyde (1565).

Several pure cultures of the fungus Trichoderma Viride were tested. Of eighteen variants, eight cultures produced DDD and kelthane from DDT; 3 produced DDD only; one produced DDD and DDE; and six were unable to degrade DDT under the test conditions used (1622).

C^{14}-labeled DDT was added to soil and the mixture was incubated anaerobically for two weeks. At that time, 10% of the radioactivity was in DDD, 88% in DDT, and less than 2% in other products: DDA; DDE; Kelthane; 4,4'-dichlorobenzophenone; 4,4'-dichlorodiphenylmethane; and p-chlorobenzoic acid. After four weeks of incubation, 62% of the radioactivity was in DDD; 34% in DDT, and 4% in other products (572). DDD formed in submerged soil (1736).

After spraying with an emulsifiable concentrate, the loss of DDT from foilage was studied. The residue fell to 40% of the initial deposit after three weeks and 4% after 11 weeks. Whereas the original spray contained only o,p'-and p,p'-DDT, o,p'- and p,p'-DDE appeared during the first week. After 11 weeks, traces of p,p'-DDD and p,p'-dichlorobenzophenone appeared. The persistence of DDD was comparable with that of DDT (618).

Under UV-irradiation crystalline DDT changed its physical and chemical properties and insecticidal activity (1430). Irradiation of p,p'-DDT at 2537A gave rise to p,p'-DDE which underwent further conversion to a number of compounds (122, 1073, 1228) including dichlorobenzophenone (462). A small portion of p,p'-DDD was dehydrochlorinated (1228).

TDE was converted to TDEE in tobacco smoke. TDE, TDEE, and DDA were found in the urine of New Zealand Red rabbits that had been exposed to cigarette smoke containing labeled TDE. In the fat, vital organs, and other tissues, TDE and TDEE were found (147).

Processing of food (437), boiling of vegetables (642), drying of milk into powder (853), or baking or frying of birds (1212) caused decomposition of DDT to DDD primarily.

In aqueous media and in the presence of reduced porphyrins, DDT was converted to DDD (1012). Iron porphyrin complexes are present in all aerobic organisms. When dilute solutions of Fe^{++} porphyrins ($Fe^{++}D$) were exposed to DDT at room temperature, the porphyrin complex was rapidly oxidized. The rate of oxidation of Fe^{++} by DDT was estimated from the slopes of concentration vs. time plots at various initial concentrations

of reactants. DDT obeyed the rate expression

$$\text{rate} = k_3\ [Fe^{++}D]^2[DDT]$$

and k_3 was found to be ca. $3 \times 10^7 1.^2/\text{mole}^2/\text{min.}$ (256).

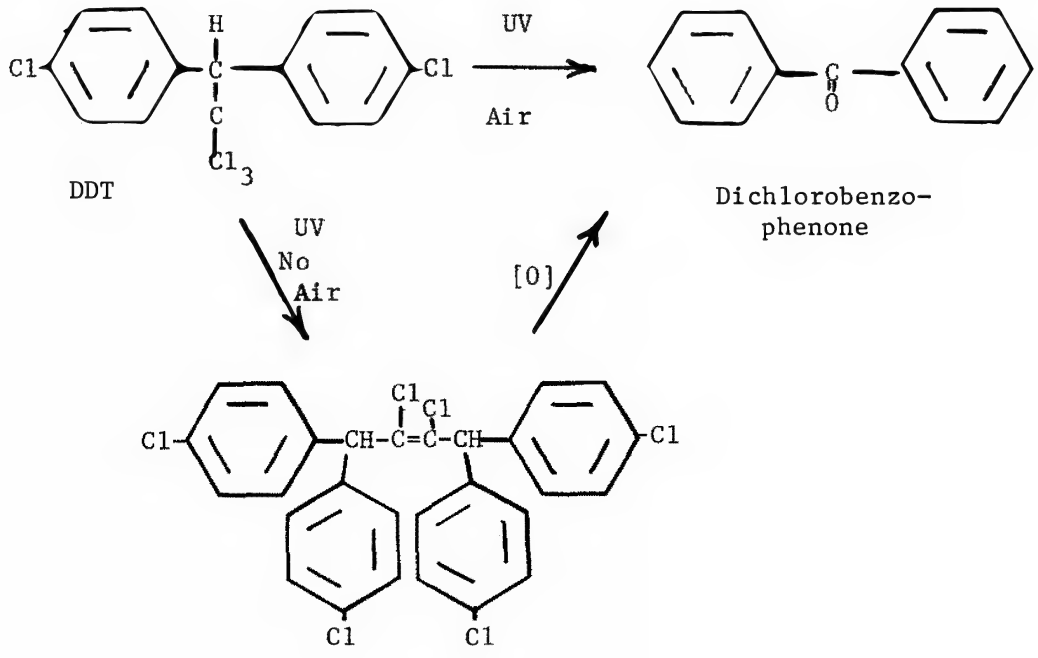
Amines also are present in all organisms; and the reaction of DDT with amines and the decomposition of DDT at room temperature was observed (898).

$$DDT + NH_2CH_3 \rightarrow DDE + CH_3NH_2 \cdot HCl$$

$$\text{Rate of reaction} = k[DDT][NH_2CH_3]^2$$

The relationship of DDT vapor pressure to temperature was determined (1615).

$$\log_{10} P_s = 9.60 - \frac{4370}{T} \quad \text{(in mmHg)}$$



DDVP (Dichlorvos Vapona) [2,2-dichlorovinyl dimethyl phosphate]

Using whole homogenates of rat and rabbit tissues, the course of metabolism of DDVP-P^{32} was followed. In liver, kidney, spleen, and adrenal glands, the principal metabolite was dimethyl phosphate (50-85%). The remainder appeared as 0-methyl 2,2-dichlorovinyl phosphate, monomethyl phosphate, and inorganic phosphate. In plasma, 98-100% of the DDVP hydrolyzed was accounted for as dimethyl phosphate. When dimethyl phosphate-P^{32} was incubated with liver homogenates or plasma, all radioactivity was recovered as unchanged dimethyl phosphate. In the presence of a rat liver soluble preparation, DDVP was degraded to dichloroacetaldehyde and some dichloroacetic acid. The aldehyde was reduced to the corresponding alcohol which was excreted as the glucuronide. Similar results were obtained with a rat, cow, and goat following oral or intraperitoneal administration (274, 678).

^{32}P-labeled DDVP was used to study dermal absorption in cows. The maximum of 0.3 ppm was reached in blood very quickly. Large amounts of ^{32}P-metabolites were immediately excreted via the urine. A part of the DDVP seemed to be reversibly bound to protein in the blood and was slowly freed. *In vitro* studies with serum indicated small amounts of desmethyl DDVP but large amounts of dimethylphosphate. In milk, the maximum level attained was 0.1 ppm; and six to eight hours post-treatment this fell to less than 0.005 ppm DDVP. Examination of the urine showed large amounts of dimethylphosphate and small amounts of desmethyl DDVP (1656).

Studies with houseflies have shown that dipterex is converted to DDVP which in turn is degraded to dimethyl phosphate and dichloroacetaldehyde (973).

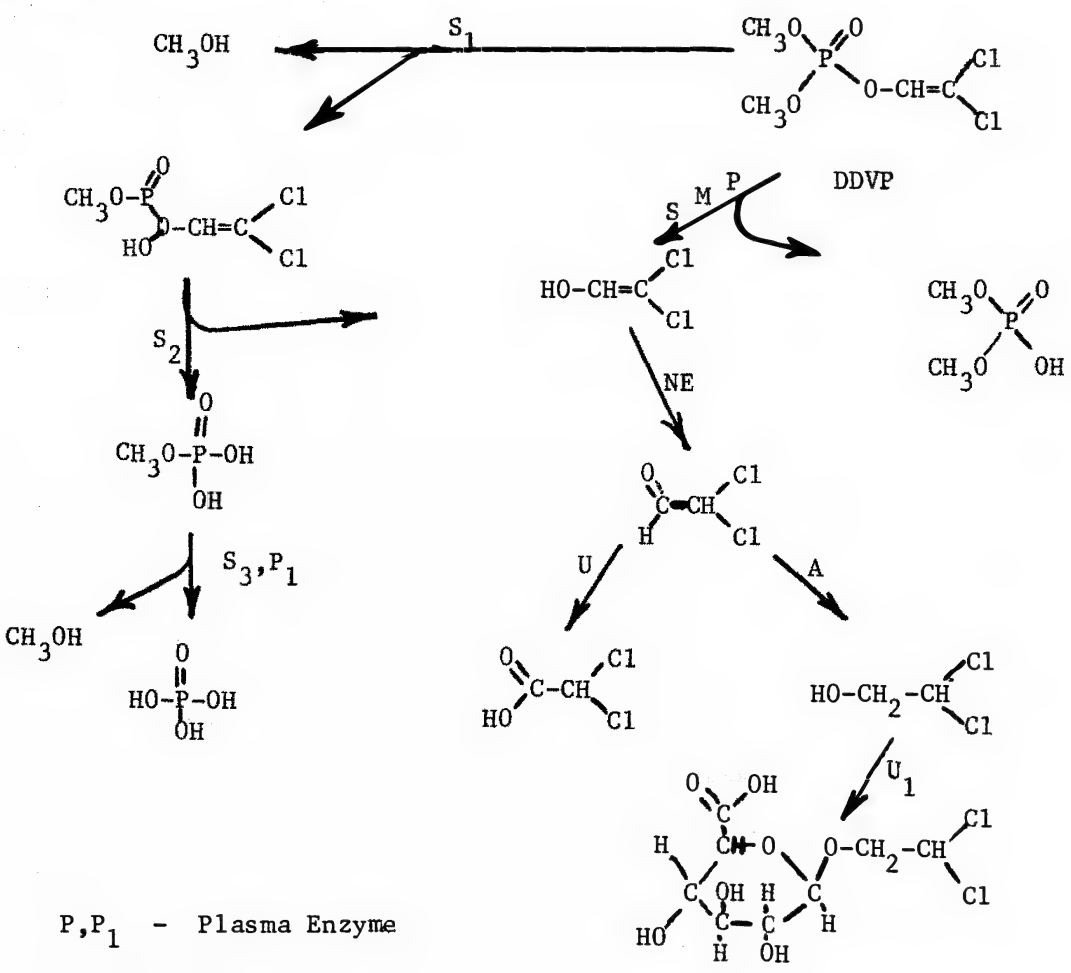
Pesudomonas melophthora and *Trichoderma viride* degraded DDVP but no metabolites were identified (145, 1622).

The half-life of DDVP at 2 ppm in aqueous solution at 38°C is 60 hours at pH=1 and 4.5 hours at pH=9.1. In saturated aqueous solution containing 1% DDVP, its hydrolysis rate is about 3% per day at room temperature. DDVP also decomposes rapidly when sorbed onto the majority of solid carriers even when the carriers have been dried (42).

After exposure of bluegill, (*Lepomis macrochirus* Rafinesque) and channel catfish (*Ictalurus punctalus* Walbaum) to DDVP, chromatography revealed the presence of dimethylphosphate and dichloroacetaldehyde (1681).

Hydrolysis

	Km (moles/l)	V (μ moles/mg Protein/hr)
Bluegill	5×10^{-2}	4.46
Catfish	5×10^{-2}	3.98



P,P_1 - Plasma Enzyme

S,S_1] - Soluble Liver

S_2,S_3] - Enzyme

M - Liver Mitochondrial Enzyme

A - Alcohol Dehydrogenase and DPNH

NE - Non-enzymatic

U,U_1 - Nature of Enzymes Not Studied

Demeton (Systox, Bayer 19639)

(I) 0,0-Diethyl S-ethyl-2-thioethyl phosphorothiolate (also known as Isosystox or Demeton S)

(II) 0,0-Diethyl 0-ethyl-2-thioethyl phosphorothioate (also known as systox thiono isomer or Demeton 0)

I $(C_2H_5O)_2-\overset{O}{\overset{\|}{P}}-S-CH_2-CH_2-S-CH_2-CH_3$

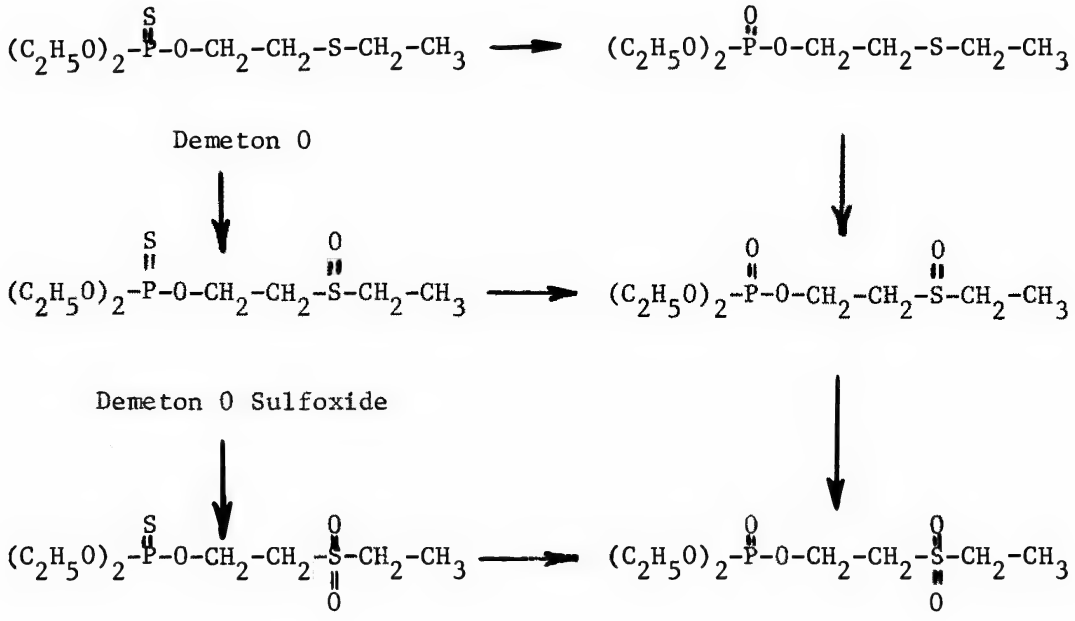
II $(C_2H_5O)_2-\overset{S}{\overset{\|}{P}}-O-CH_2-CH_2-S-CH_2-CH_3$

Investigations of the two isomers have been carried out by use of P^{32} radiotracers, by chromatography, and by infrared spectrophotometry. The data obtained showed that these isomers were metabolized in the same manner in fruits, beans, and cotton. Seven days after application of demeton 0, only traces of the isomer were present. All radioactivity on the chromatograms corresponded to the sulfoxide and sulfone (506, 746, 999, 1000, 1084). Demeton 0 also underwent isomerization in plants and gave rise to three unidentified compounds (620, 646, 1050). Up to 14 days after the topical application of demeton S to cotton, this isomer accumulated rapidly in the leaves. Subsequently, the sulfoxide analog appeared in greater quantity. Some sulfone was also observed (994).

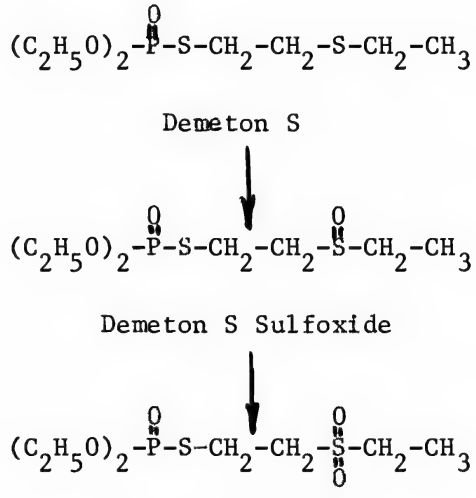
With mice, the administered dose was rapidly absorbed, metabolized, and eliminated. Only 6% or less was found in the stomach after 1 to 2 hours; and, after 4 hours, it had almost entirely disappeared from these tissues. Small amounts passed through the intestines but most was eliminated in the urine rather than the feces. The major site of degradation was the liver and degradation continued until elimination in the urine (924).

In gross aspects, the biochemical mechanisms in mice, cockroaches, and plants were similar. Major differences were concerned with rates of metabolism and degradation; and, as expected, rates were greater in the mammal than in the insect and greater in the insect than in plants. The routes of metabolism and the metabolites, however, were the same in each case. The principal metabolic path for both isomers was the oxidation of the 2-ethylthio ether to the sulfoxide and sulfone. In the case of demeton 0, a secondary path involved oxidation of P=S to P=0 with subsequent oxidation to the sulfoxide and sulfone (924).

Investigations with demeton-S have shown that thin films undergo rapid conversion to more hydrophilic compounds when exposed to air and light. Following application to plants, demeton-S rapidly gave rise to at least three metabolites, two of which persisted for as long as 15 weeks. Using chromatographic techniques, two of the compounds were identified as the corresponding sulfoxide and sulfone (504, 639, 1402, 1404, 1405).



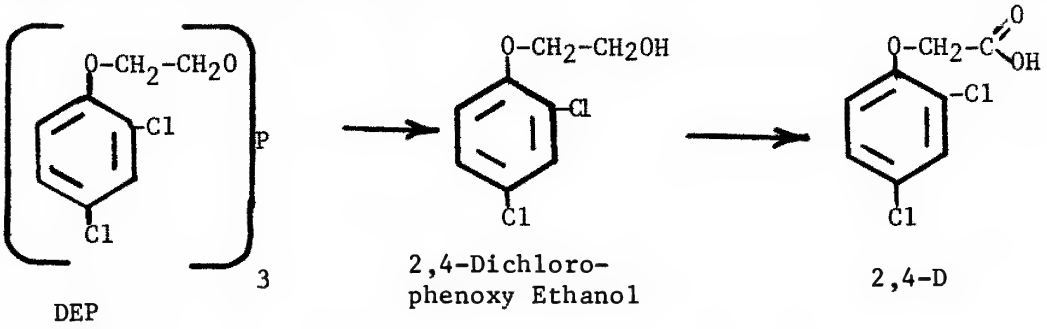
Demeton O Sulfone



Demeton S Sulfone

DEP (Falone)[2,4-Dichlorophenoxyethyl phosphite]

In the presence of moisture, DEP underwent hydrolysis with formation of inorganic phosphate and 2,4-dichlorophenoxyethanol. This could then undergo oxidation to 2,4-D (44, 1288).



DEP

2,4-Dichloro-phenoxy Ethanol

2,4-D

Dexon [(p-Dimethylaminobenzenediazo sodium sulfonate)]

Rhizoctonia and sugar beets have a mitochondrial system that decomposes dexon in the presence of DPNH (1410). Studies with C^{14}-labeled dexon and Aphanomyces cochloides Drechs. showed that it was taken up by roots and translocated to hypocotyls. Much of the label was not extractable in aqueous methanol. Radioautographs of root extracts showed that dexon was rapidly converted into at least four unidentified C^{14}-labeled compounds (657).

Studies with aqueous solutions of dexon suggested a two stage decomposition in the presence of light and air: (1) photochemical reduction of the diazonium group and evolution of nitrogen; (2) oxidation followed by polymerization of the reaction products. During these developments, the solution changes in color from yellow to pink and deepens to purple. Spectrophotometric studies indicated that any p-dimethylaminophenol formed was rapidly converted to other compounds (658).

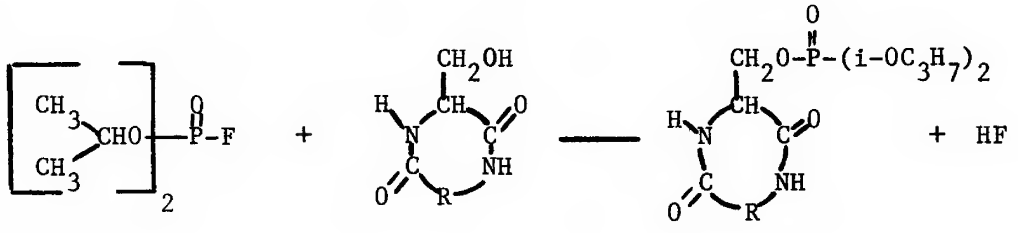
Studies showed that a DFPase was present in mammals and that it was capable of splitting the P-F bond and releasing HF. Phosphorylation of the serine moiety of the enzyme occurred simultaneously (51, 52, 719, 789, 949, 1046, 1047, 1048, 1064, 1581).

When small amounts (0.1 mg/kg) of DFP were administered to guinea pigs, DFP was preferentially bound by serum and by the lungs. Administration of large amounts (3-6 mg/kg) produced uniform body distribution and accumulation in kidneys and liver. DFP was converted in part to diisopropyl phosphate (DP). Both DFP and DP were excreted mainly in the urine. Some appeared in bile (293, 612).

DFP was degraded by Pseudomonas melophthora but no metabolites were identified (145).

Bluegill (Lepomis macrochirus Rafinesque) and channel catfish (Ictalurus punctalus Walbaum) converted DFP to diisopropyl phosphate (1681).

	Hydrolysis	
	Km (moles/l)	V(μ moles/mg protein/hr)
Bluegill	3×10^{-3}	13.62
Catfish	5×10^{-3}	9.72



DFP

Diazinon [0,0-diethyl O-(2-isopropyl-4-methyl-6-pyrimidinyl) phosphorothioate]

Studies with mice and insects have been conducted. The only metabolite identified was the P = O analog (466, 596, 840). In cows, the diethyl-phosphorothioic and phosphoric acids were found (1220).

Fish (Fundulus heteroclitus L.) and freshwater mussels (Elliptio complanatus Solander) were exposed to diazinon-C^{14} contaminated waters. Only diazinon was detected in the fish. It was assumed that some of the labeled portion was split off and metabolized to $C^{14}O_2$ (1006).

Tomato plants grown in a fenced and controlled plot were sprayed with S^{35}-diazinon. The only metabolite identified was oxo-diazinon (diazoxon). After spraying with C^{14}-diazinon, 2-isopropyl-4-methylpyrimidin-6-ol was found. The formation of radioactive barium carbonate was evidence that the pyrimidine ring of diazinon was degraded by tomato plants (1149, 1616). In wheat grain and bean plants (Phaseolus vulgaris) treated with diazinon, small amounts of 2-isopropyl-4-methylpyrimidin-6-ol and unextractable metabolites were observed. Diazinon was not found after 30 days (756, 1573). On treated spinach, high residue levels of the pyrimidinol and oxon metabolites were observed (1616). An alteration product of diazinon was isolated from field sprayed kale that exhibited retention times on two GLC columns and an R_f value on TLC identical with those of 0,0-diethyl-0[2-(2'-hydroxy-2'-propyl)-4-methyl-6-pyrimidinyl]phosphorothioate, a compound prepared by ultraviolet irradiation of diazinon. Tentative identification was made by infrared and mass spectroscopy (1646).

Pseudomonas melophthora and Trichoderma viride degraded diazinon but no metabolites were identified (145, 1622). When diazinon was incubated with streptomyces or arthrobacter individually, the pyrimidyl ring was not attacked; however, when these two organisms were incubated together, they metabolized the ring of diazinon. Two metabolites were observed but not identified (576).

In soil, hydrolysis was a major mechanism in diazinon degradation (822). Three weeks after treatment of Sultan silt loam with diazinon, in addition to diazinon, 2-isopropyl-4-methyl-6-hydroxypyrimidine was identified. The oxon analog was unstable in soil and was not detected. When C^{14}-ring-labeled diazinon was used, $C^{14}O_2$ was produced (542).

The effect of soil microorganisms, moisture and type and diazinon concentration on the disappearance rate of diazinon was studied. Results indicated that all four factors influenced the degradation rate, microorganisms having the greatest effect and diazinon concentration the least. Diazinon half-life varied from 21.6 days to 80.2 days (1555).

Hydrolysis of diazinon and diazoxon conform to a pseudo first-order rate in the pH range 3.1 to 10.9. Diazinon hydrolysis was rapid under acid conditions. At neutral or alkaline pH, the rate was lower. Diazoxon hydrolysis proceeded much more rapidly than diazinon. Using IR and UV with gas-liquid chromatography, 2-isopropyl-4-methyl-6-hydroxypyrimidine was identified as the major hydrolysis product (1637).

Diazinon

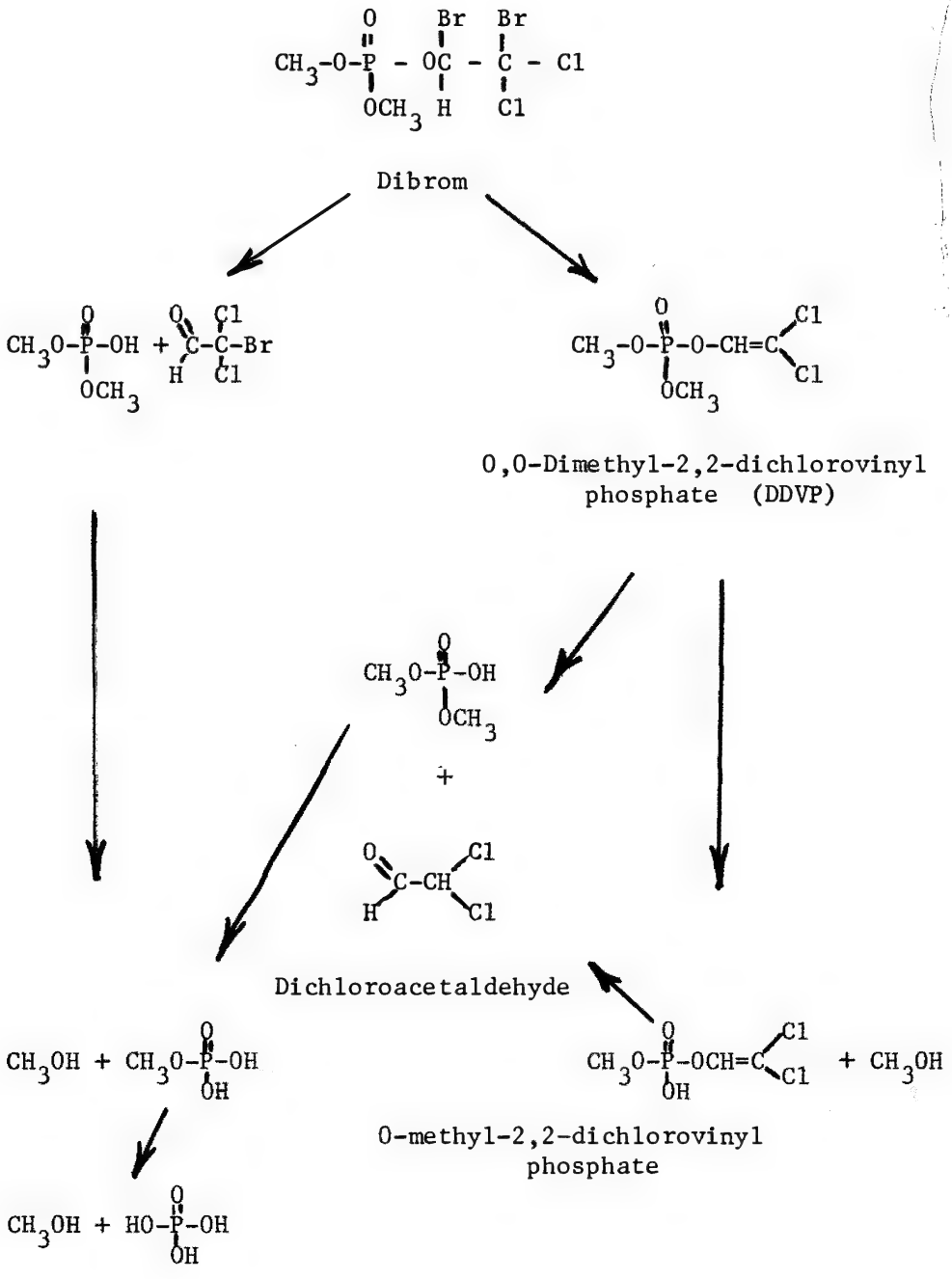
pH	t 1/2
3.14	706 minutes
7.4	4435.8 hours
9.0	3263.0 hours
10.9	144.9 hours

Sodium azide caused the disappearance of diazinon from distilled water by catalyzing its hydrolysis into diethylphosphorothioic acid, 2-isopropyl-4-methyl-6-hydroxypyrimidine and three other compounds not identified (1644).

Microsomes, prepared from the livers of adult male rats, in the presence of $NADPH_2$, converted diazinon to diethyl phosphorothioate and phosphate and diazixin (1782).

Dibrom (1,2 dibromo -2,2-dichloroethyl dimethyl phosphate)

Adult male rats were treated with dibrom-P^{32} in corn oil by stomach tube. Within a day, high activity appeared in the bone. After oral administration of dibrom-P^{32} to a cow, urine yielded P^{32}-metabolites chromatographing in the position of inorganic phosphate, mono- and dimethyl phosphate, and O-methyl 2,2-dichlorovinyl phosphate, DDVP, dichloroacetaldehyde, dichlorobromoacetaldehyde. Studies indicated that dibrom reacts with sulfhydryls and that certain complex amino acid conjugates of the degraded products are also formed (247, 813).


Dibrom
0,0-Dimethyl-2,2-dichlorovinyl phosphate (DDVP)
Dichloroacetaldehyde
0-methyl-2,2-dichlorovinyl phosphate

Dicamba (Banvel D) [2-Methoxy-3,6-dichlorobenzoic acid]

Dicamba was rapidly excreted by rats after injection or oral administration, unchanged and as the glucuronic acid conjugate (1437).

The metabolism of dicamba by wheat (var. Gaines) and bluegrass (var. Newport) plants yielded three metabolites. Paper chromatography was used to separate and detect the metabolites. Gas liquid chromatography, infrared and mass spectrometry were used to identify the major (90%) metabolite as 5-hydroxy-2-methoxy-3,6-dichlorobenzoic acid. After hydrolysis by β-glucosidase, one of the minor (5%) residues was identified as dicamba. The second minor (5%) metabolite was identified as 3,6-dichlorobenzoic acid (182).

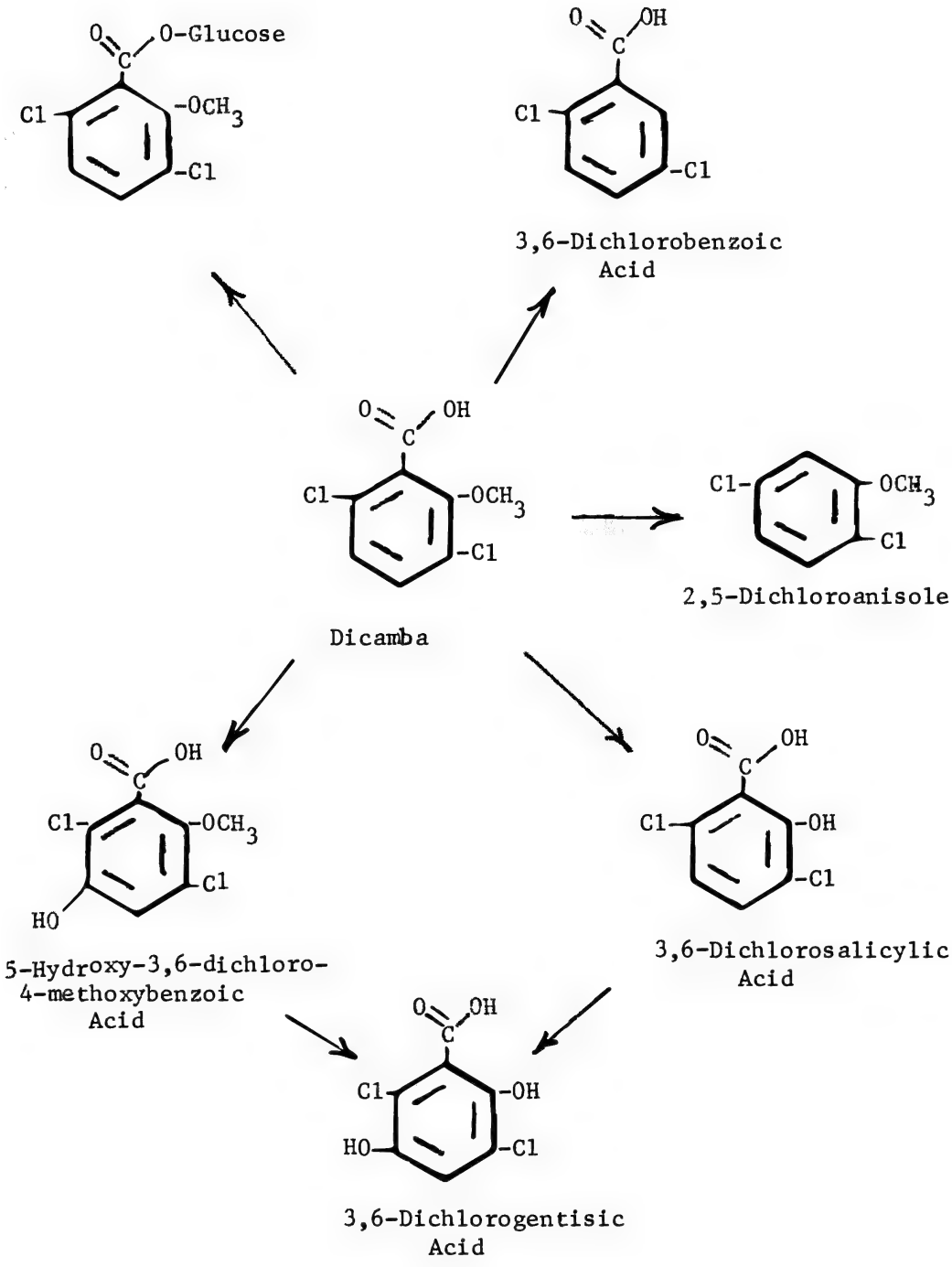
Roots of plants which had been incubated with dicamba were homogenized and analyzed. Gas chromatography of propyl and butyl derivatives showed that 5-hydroxy-dicamba was a major metabolite in treated corn roots and that 3,6-dichlorosalicylic acid was a minor metabolite in treated roots of both corn (Zea) and barley (Hordeum). The data also indicated that 5-hydroxy-dicamba and 3,6-dichlorogentisic acid were major metabolites in young barley plants treated with dicamba (1196).

Studies have shown that microbial or chemical decomposition of dicamba in soil was more important that volatilization (209).

Dicamba moved readily in soil water, suggesting limited adsorption (498).

Studies have indicated that dicamba exerted its effects by inhibiting pantothenate synthesis at the level of conversion of ketopantoate to pantoate (659).

At 200°C dicamba decarboxylated to give 2,5-dichloroanisole (1761).


O-Glucose
Cl
-OCH3
-Cl
OH
3,6-Dichlorobenzoic Acid
2,5-Dichloroanisole
Dicamba
5-Hydroxy-3,6-dichloro-4-methoxybenzoic Acid
3,6-Dichlorosalicylic Acid
HO
3,6-Dichlorogentisic Acid

Dicapthon (Dicaptan, Isomeric Chlorothion) [0-(2-chloro-4-nitrophenyl) 0,0-dimethyl phosphorothioate]

Rabbit and rat liver microsomes and housefly microsomes plus $NADPH_2$ and O_2 were incubated with dicapthon In each case, the corresponding phenol was found. Activation of dicapthon to the corresponding oxon analog was observed. Some conversion of the oxon analog to the corresponding phenol was also observed (1700).

Ditholane (EI-43064; Cyolane) [2-(diethoxyphosphinothioylimino)-1,3-dithiolane]

No metabolites were identified or demonstrated. Differential cholinesterase inhibition was cited as evidence of degradation to the oxon analog (1739).

Dichlobenil (Casoron) [2,6-Dichlorobenzonitrile]

Prefix [2,6-Dichlorobenzenethioacetamide] Chlorthiamid

Dogs (beagles) and rats (Portan strain) did not show species differences in the patterns of elimination of metabolites of either Prefix or Dichlobenil. Single labeled doses of either compound were almost entirely eliminated in four days. Less than one-half of 1% of the dose was present in the carcass and viscera after removal of the gut. Most of the metabolites, upwards of 60%, appeared in the urine. In the unhydrolyzed urine of rats dosed with either Prefix or Dichlobenil, free and conjugated metabolites were found: 2,6-dichloro-3-hydroxybenzonitrile and 2,6-dichloro-4-hydroxybenzonitrile and their glucuronides and sulfate, and the ester glucuronides of 2,6-dichlorobenzoic acid and 2,6-Cl_2-3-hydroxybenzoic acid. An unknown aromatic acid and two sulfur containing metabolites--possibly mercapturic compounds--were also observed (564).

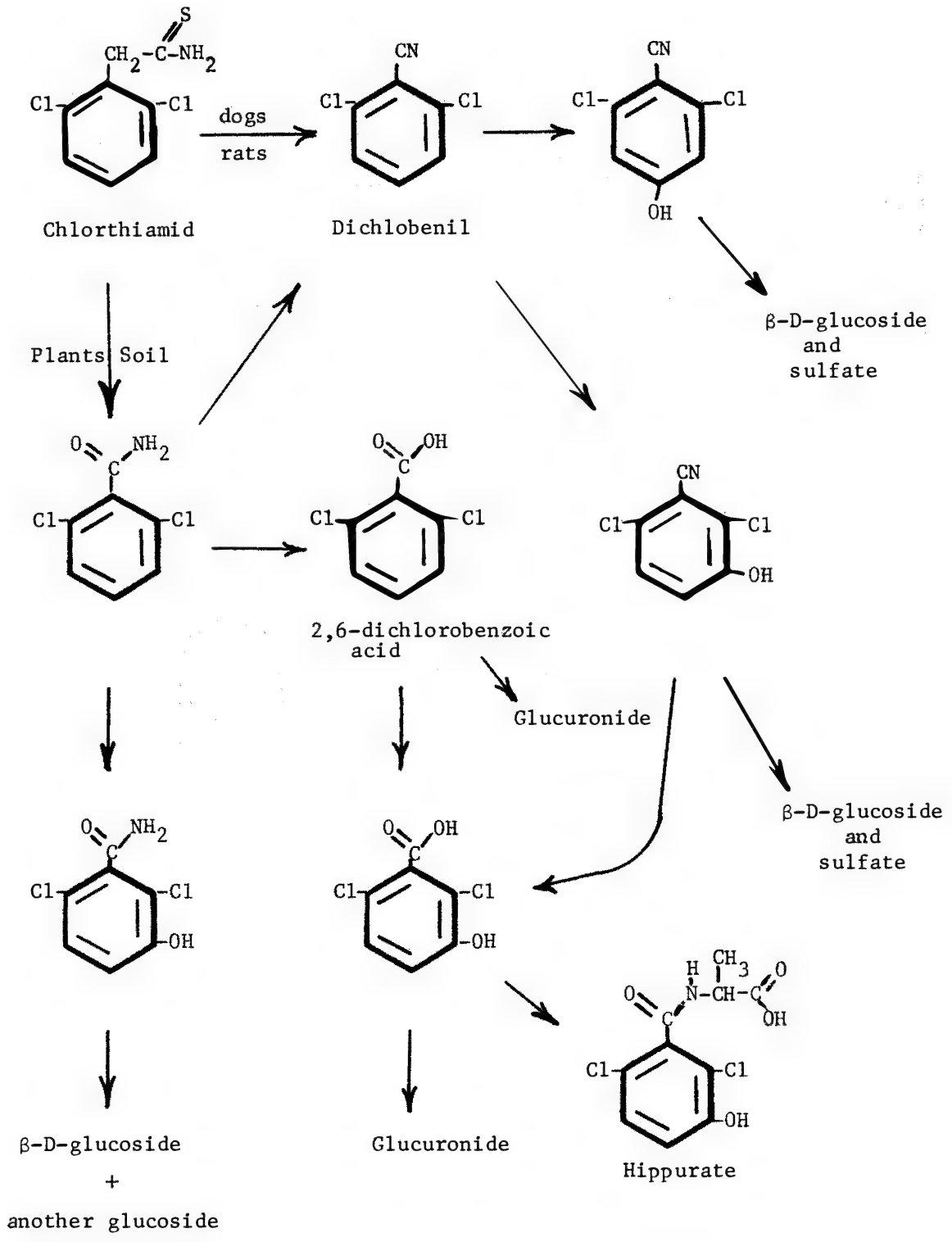
Examination of the collected urine of rabbits and rats fed dichlobenil showed the presence of residues in the amount of 2% and 6%, respectively, for these animals in the form of the 4-hydroxy analog; and 23% and 22%, respectively, in the form of 3-hydroxy analog. The hippurate conjugate was also observed (1517).

2,6-dichlorobenzonitrile was found to be the main metabolite of Prefix in plants and unsterilized soil. There was slight evidence that Prefix also gave rise to a limited extent to the corresponding amide and thence to 2,6-dichlorobenzoic acid (564).

Exposure to labeled dichlobenil showed that this compound can be absorbed from the atomosphere as well as from an aqueous solution. Alligatorweed [Alternanthera philoxeroides (Mart.) (Griseb.)], bean (Phaseolus vulgaris L.), fungi (Fusarium sp., Geotrichum sp., Penicillium sp., and Trichoderma sp.) and a mematode (Rhabditis sp.) were used to study dichlobenil metabolism. From water extracts, 2,6-dichlorobenzoic acid was isolated and identified by autoradiographs of thin-layer chromatograms and gas chromatography (932, 1133, 1134, 1135, 1136).

When exposed to ultraviolet light, dichlobenil underwent dechlorination (1170).

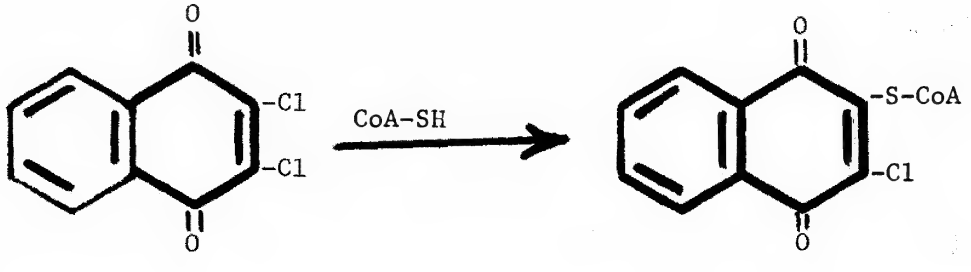
Rice, wheat (Rothwell Perdix), and apples (Ellison's Orange) were grown in a glasshouse in soil treated with ^{14}C-chlorothiamid (Prefix). Dichlorobenzonitrile and the benzamide were found in the soil, rice stems and leaves, and apple leaves. The benzamide was also found in wheat grain, straw and leaves. In extracts of wheat, 3-hydroxy-2,6-dichlorobenzamide was identified both before and after treatment with β-glucosidase. After treatment of apple leaves with ^{14}C-2,6-dichlorobenzamide, the glycosidic material was shown to contain two components. One was identified as the β-D-glucoside of 3-hydroxy-2,6-dichlorobenzamid. The other was a glycoside of the same aglycone with a sugar other than β-D-glucose. The 3-hydroxy- and 4-hydroxy-2,6-dichlorobenzontriles were identified in apple leaf and wheat leaf after enzymic hydrolysis (1765, 1766, 1767).


Chlorthiamid
dogs
rats
Dichlobenil
β-D-glucoside
and
sulfate
Plants Soil
2,6-dichlorobenzoic
acid
Glucuronide
β-D-glucoside
and
sulfate
β-D-glucoside
+
another glucoside
Glucuronide
Hippurate

Dichlone (Phygon) [2,3-Dichloro-1,4-naphthoquinone]

Experiments indicated that the key oxidations of glucose, acetate, pyruvate, and α-ketoglutarate were inhibited by dichlone. Since coenzyme A is the only factor common to all these reactions, the data strongly indicated that coenzyme A was inactivated. Other studies have also shown that dichlone inhibits enzymes with free sulfhydryl groups. Whether subsequent degradation of the CoA complex occurs is not now known (962, 1121, 1122).

Under ultraviolet light, dichlone reacted with pyrid ne nucleotides and underwent reduction (1547).



Dichloran (Botran, Ditranil, Allisan, DCNA) [2,6-Dichloro-4-nitroaniline]

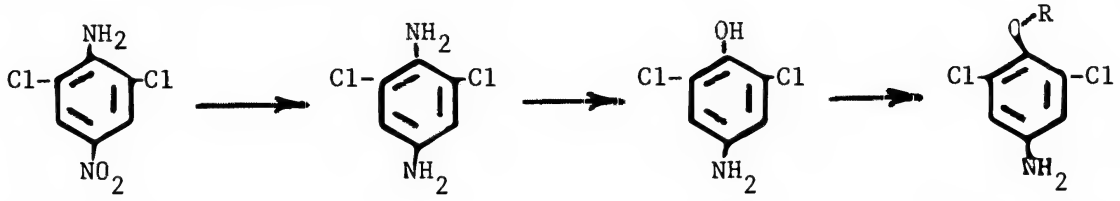
After oral administration of C^{14}-labeled dichloran to male rats, Dichloran was rapidly absorbed, metabolized, and excreted with 89% of the label in the urine. Urinary excretion was first order, k=0.199/hr. and $T_{1/2}$ = 3.5 hours. Twenty-four hour urine pools contained only free and conjugated 2,6-dichloro-4-hydroxyaniline (DCHA); 1 dichloran: 1 dichloran glucuronide: 6 dichloran sulfate. Hydrolysis of the urine showed two compounds in addition to traces of starting material. 4-amino-3,5-dichlorophenol accounted for 70% of the urinary radioactivity; 4-amino-2,6-dichloroaniline for about 2.4%. Small amounts of an aryldiamine was indicated (392, 393).

In vitro studies showed that mouse liver microsomes converted dichloran to the aminophenol and the phenylenediamine. TLC showed that this conversion was small. About 91% of the Botran was recovered unchanged (393, 934).

Excretion of urinary radioactivity in human subjects could be described by two simultaneous pseudo 1st-order process with mean rate constants of 0.0749 and 0.0260/hour, which are equivalent to excretion half-times of 9.26 and 27.4 hours, respectively (392).

Dichloran was strongly fixed in clay soils and was not completely extracted from treated soils. It was slightly water soluble and leached slowly (571).

Dichloro-4-nitroaniline (DCNA) was readily taken up and translocated by both tomato and lettuce and was rapidly degraded to unidentified polar metabolites. Labeled carbon from DCNA-C^{14} appeared in the carbohydrate plant constituents (861).



Dichloran

DCHA (4-Amino-3,5-Dichlorophenol)

Glucuronide and Sulfate

Dichlorobenzene (DCB)

o-Dichlorobenzene (o-DCB)

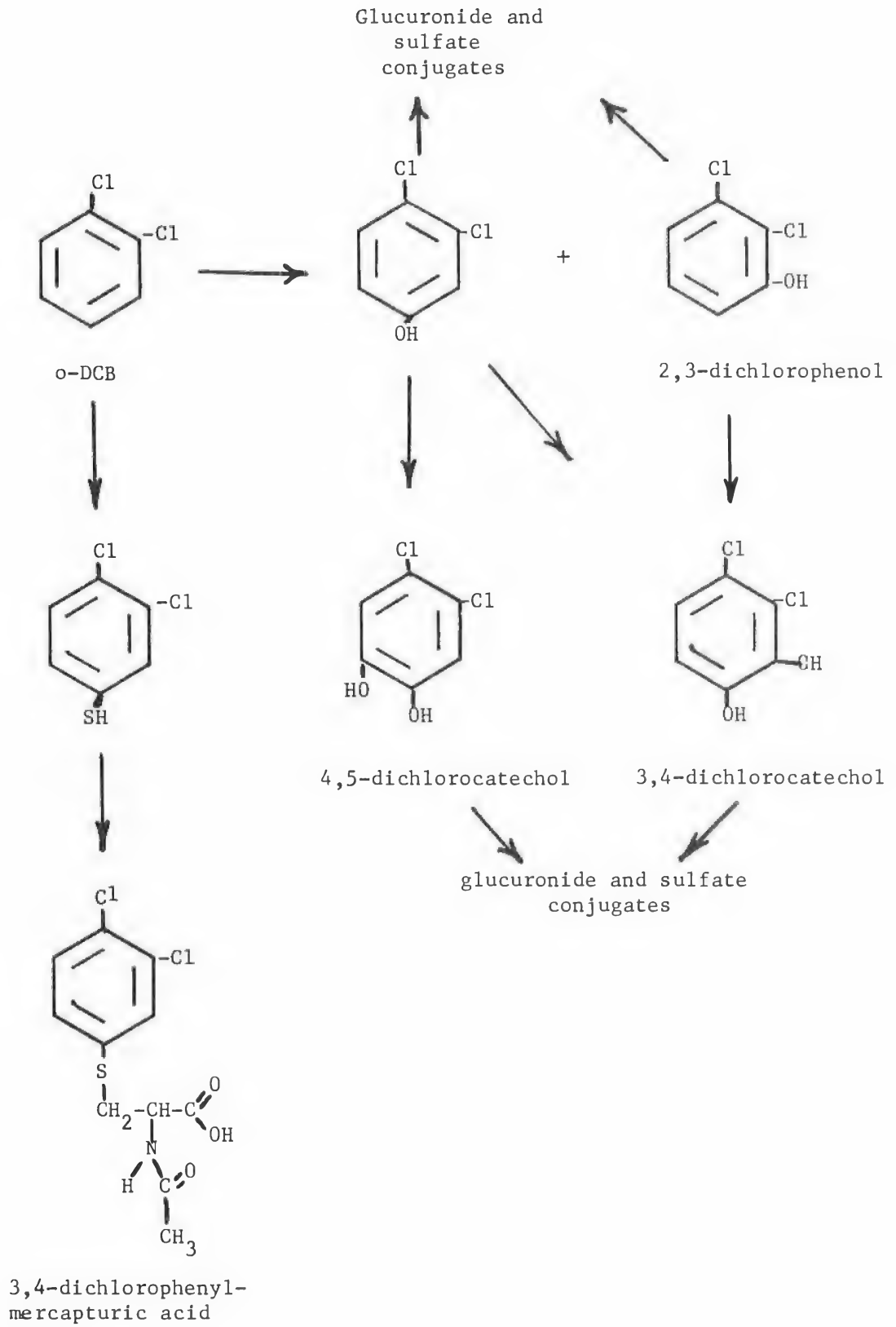
After ingestion of o-dichlorobenzene, urine of rabbits were found to contain 2,3- and 3,4-dichlorophenol free and as O-glucuronic and sulfuric acid conjugates, 3,4- and 4,5-dichlorocatechol free and as the O-glucuronide and sulfate, and 3,4-dichlorophenylmercapturic acid (56, 57).

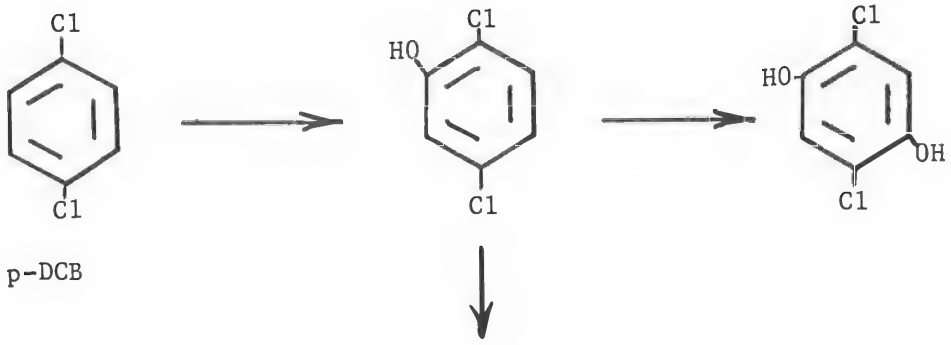
p-Dichlorobenzene (p-DCB)

After ingestion of p-dichlorobenzene, 2,5-dichlorophenol (30%) free and as the glucuronide and sulfate and 2,5-dichloroquinol (6%) were excreted (57). In humans, 2,5-dichlorophenol was also found in the urine (1124).

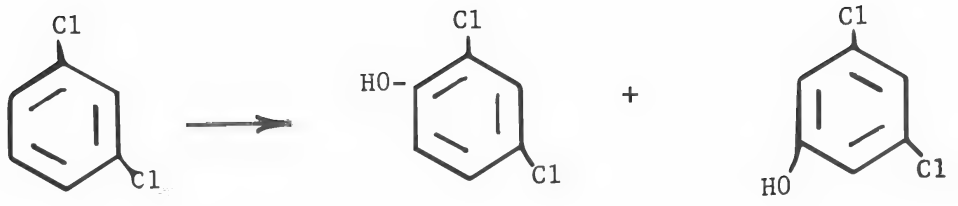
m-Dichlorobenzene (m-DCB)

Urine was collected from a rabbit fed m-dichlorobenzene. Chromatography of the monophenolic fraction revealed the presence of 2,4- and 3,5-dichlorophenol. In the dihydric phenol fraction, 3,5-dichlorocatechol and 4-chlorocatechol were found. A mercapturic acid observed was identified as 2,4-dichlorophenylmercapturic acid (1126, 1127).


Glucuronide and sulfate conjugates
Cl
Cl
Cl
OH
+
Cl
Cl
OH
o-DCB
2,3-dichlorophenol
Cl
Cl
SH
Cl
Cl
HO
OH
Cl
Cl
OH
4,5-dichlorocatechol
3,4-dichlorocatechol
glucuronide and sulfate conjugates
Cl
Cl
S
CH2-CH-C
O
OH
N
H
C
O
CH3
3,4-dichlorophenyl-mercapturic acid



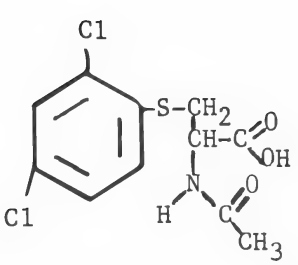
glucuronide
and
sulfate

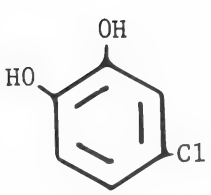


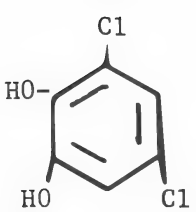
m-DCB

2,4-
Dichlorophenol

3,5-
Dichlorophenol







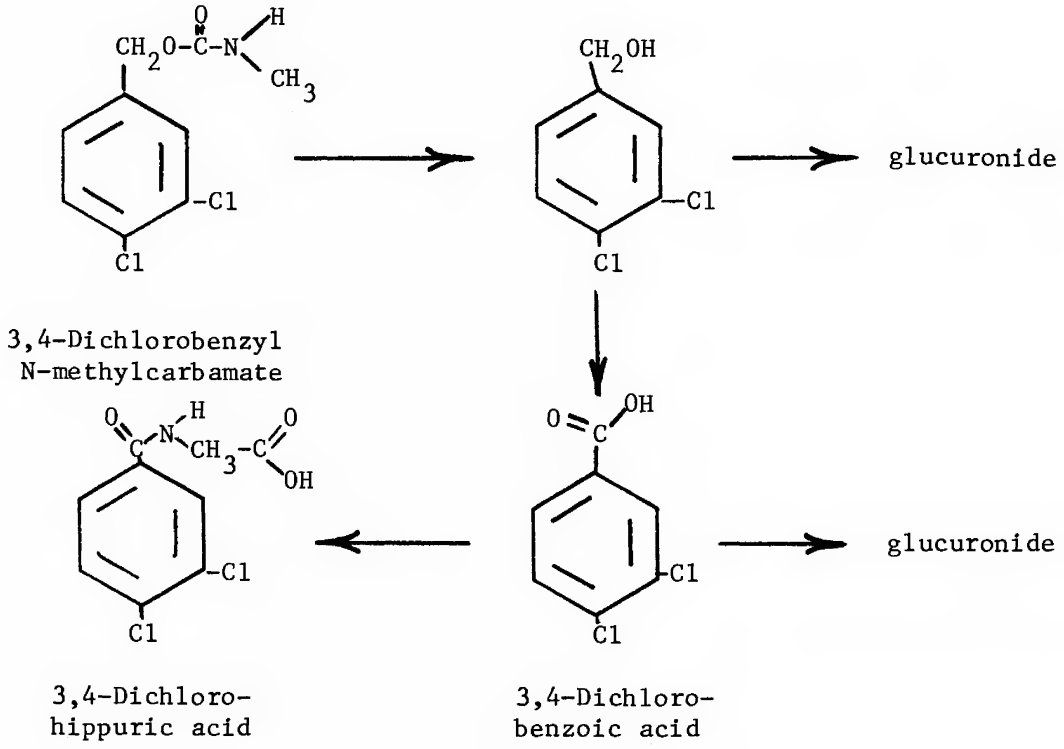
2,4-dichloro
mercapturic acid

4-Chlorocatechol

3,5-dichlorocatechol

Rats treated with labeled 3,4-dichlorobenzyl N-methylcarbamate excreted the labeled material in feces and urine and in expired CO_2. Tissue residues were observed only in the case of methyl labeled carbamate. Using gas chromatography, 3,4-dichlorohippuric acid (63%), 3,4-dichlorobenzoic acid (6.22%), 3,4-dichlorobenzyl glucuronide (5%) and what was thought to be 3,4-dichlorobenzoyl glucuronide were observed (1371, 1576).

In plants, 3,4-dichlorobenzyl N-methylcarbamate was degraded to several unidentified compounds (653).

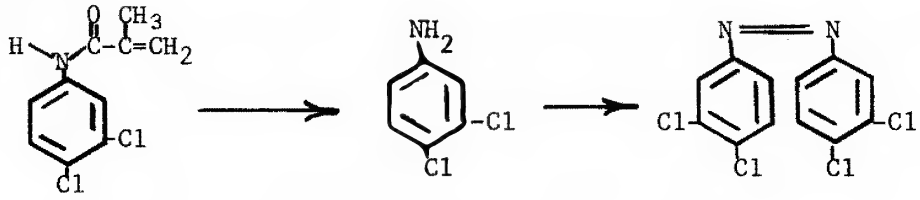


1,3-Dichloropropene, cis- and trans-

Cis- and trans- 1,3-dichloropropene are hydrolyzed in wet soil to the corresponding allyl alcohol (257).

Dicryl [N-(3,4-dichlorophenyl)-methacrylamide]

Soil samples were treated with Dicryl and incubated for two weeks. Gas chromatography, thin-layer chromatography, melting points, and infrared were used to identify metabolites in the residues as dichloroaniline and tetrachloroazobenzene (1627). (See also Dichloran, Karsil, Propanil and Ramrod)



Dimefox (DMF) [bis(dimethylamido)phosphoryl fluoride]

Although it is known that dimefox is converted to an anticholinesterase in the liver of rats and in the gut of roaches, no metabolites have been identified. By analogy to schradan, the N-oxide can be postulated, (34, 446).

Dimethoate (Rogor) [0,0-Dimethyl S-(N-methylacetamide) phosphorodithioate]

Rats degraded dimethoate to the oxygen analog, the carboxy derivative, desmethyl dimethoate, the 0,0-dimethyl-phosphoric, phosphorothioic, and phosphorodithioic acids. Several other compounds were unidentified. When the carboxy derivative was administered, 0,0-dimethyl phosphorodithioic, 0,0-dimethyl phosphorothioic, and 0,0-dimethyl phosphoric acids were found. Highest levels of radioactivity persisted in the liver, skin, and bone. Dimethoate residues were present as long as 672 hours after administration (167, 339, 840, 1168, 1266, 1438). In studies with rat liver homogenates, desmethyl dimethoate, the S-acetic acid analog, dimethyl phosphorothioate and dimethyl phosphorodithioate were identified (1039). Mice, exposed to dimethoate, excreted mono- and di-methyl phosphate and di-phosphate and orthophosphate (1079).

Dimethoate was applied dermally to a lactating cow. Thin-layer chromatography indicated the presence, in addition to dimethoate, of dimethoxon, dimethoate acid and desmethyl dimethoate in the blood; and of dimethoxon, dimethylthio (or dithio) phosphate and possibly desmethyl dimethoate in the milk. Several additional materials were not observed (359).

From urine samples from a steer treated with labeled dimethoate, the following metabolites were isolated and identified: 0,0-dimethyl S-carboxymethyl phosphorodithioate, 0-methyl S-(N-methylcarbamoylmethyl) phosphorothioic acid, 0,0-dimethyl phosphoric acid, 0,0-dimethyl phosphorothioic acid, and 0,0-dimethyl phosphorodithioic acid. Of 34 tissues tested for residue levels, liver and kidney contained the greatest amount of total dimethoate equivalents and of actual dimethoate (339, 758). Similar results were obtained with sheep (265). Other mammalian studies have indicated that the predominant path was dependent on species, sex and concentration of the dimethoate.

In the pheasant, the toxic thiolate analog tends to accumulate and its conversion to one of the other metabolites is much slower than in mammals. This probably explains the greater susceptibility of the pheasant (1266).

When P^{32}-labeled dimethoate was applied to bollworm larvae (Heliothis zea) and adult boll weevils (Anthonomus grandis), metabolic products were similar to those reported for mammals (203). From internal extracts of topically treated boll weevils, seven metabolites were isolated and identified. Among these were dithioate, and the thiocarboxy and oxygen analogs of dimethoate, and what was believed to be the oxycarboxy analog. Bollworms were treated by injection of labeled dimethoate. The highly toxic oxygen analog was highest in concentration. Later, considerable amounts of dithioate were found. The metabolite of major imporance which was found was dimethyl

phosphate. This accumulated and degraded slowly. The desmethyl carboxy derivative and what was thought to be monomethyl phosphate were also present (1266). In the cotton leafworm (Prodenia Litura F.), dimethoate gave rise to the oxygen analog, carboxy derivative, dimethylphosphorothioate, dimethyl phosphorodithioate, mono- and di-methyl phosphate, orthophosphate and thiophosphate (1636).

Metabolism of dimethoate by the American cockroach (Periplaneta americana L.), the German cockroach (Blatella germanica L.), and the house fly (Musca domestica L.) was qualitatively similar to that of rats. Quantitative differences were observed (167, 1039).

Studies have been conducted with other insects. In the locust, the main product was the highly toxic thiolate analog (dimethoxon) (1266). Using the green rice leafhopper (Nephotettix cincticeps Uhler), the mono- and di-methylphosphates and orthophosphate were observed. Homogenates of houseflies, 4th to 5th instar larvae of rice stem borer (Chilo Suppressalis Walk.), adults of apterous form of green peach aphid (Myzus persicae Sulzer) and adult male American cockroaches degraded dimethoate. Desmethyl dimethoate, the carboxy acid analog and dimethyl phosphorothioate were identified (1039).

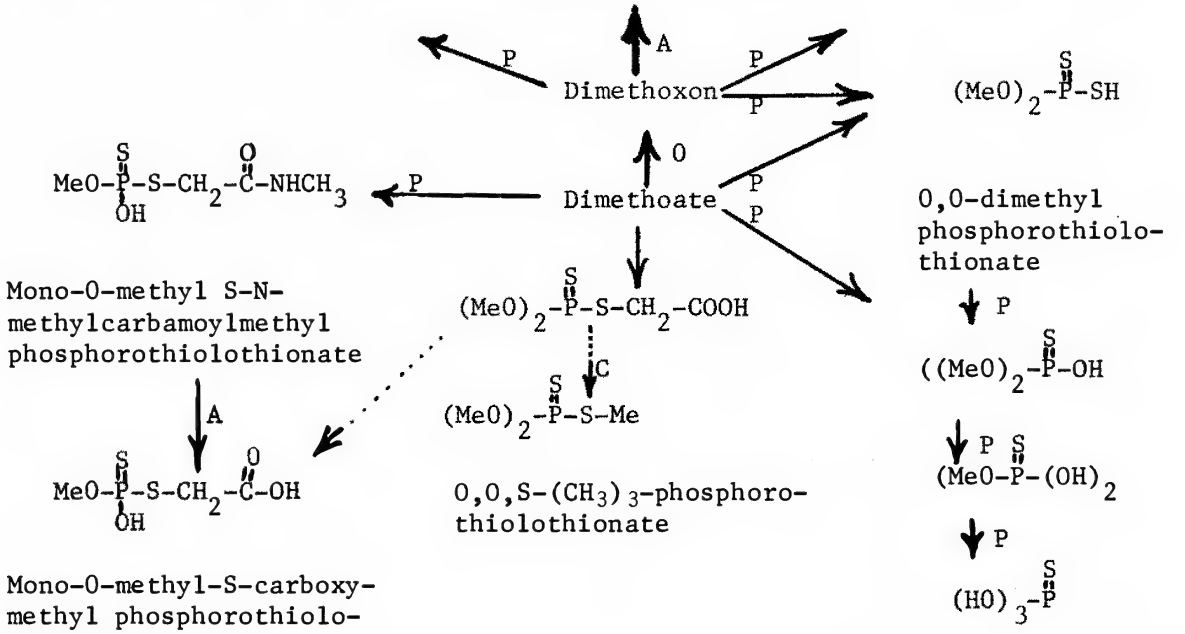
When applied to plants, dimethoate was rapidly absorbed and decomposed both on the surface and within the plant by hydrolysis and oxidation. On the plant surface, dimethoate underwent non-enzymatic oxidation to the oxygen analog and hydrolysis to water soluble derivatives identified as dimethyl phosphoric and 0,0-dimethyl phosphorothioic acids, desmethyl dimethoate, and a compound thought to be the oxycarboxy derivative. Within plants, the oxygen, desmethyl, thiocarboxy and oxycarboxy analogs, monomethyl and dimethyl dithioate, monomethyl and dimethyl thioate, dimethyl phosphate and phosphoric acid were found. Inside the plant the desmethyl analog was predominant, whereas, on the surface the oxycarboxy compound predominated. Marked quantitative differences found in the levels of the different water soluble derivatives in and on corn, cotton, olives, sugar beets, cucumbers, and potato plants indicated different paths of degradation (341, 597, 842, 1107, 1268, 1269, 1270, 1271, 1272).

In a pot test, P^{32}-dimethoate was applied to onions. After six days only dimethoate and $(CH_3O)_2P(O)SCH_2CONHCH_3$ (in a 6:1 ratio) were observed (1107). Rice and apple leaves degraded dimethoate to desmethyl dimethoate, dimethyl phosphate and orthophosphate. With in vitro studies, cabbage leaves gave rise to desmethyl dimethoate, the carboxy acid analog and dimethyl phosphate; with rice leaves, the desmethyl analog was identified (1039).

An investigation was carried out to see whether oil obtained from dimethoate-treated olives contained any decomposition products of dimethoate. Although the oxon analog was present in the olives, none was found in the oil. (14, 1261, 1262).

Oxidation of dimethoate to its thiolate analog occurred in seed coats and germ of wheat grains. Dicarboxylase activity was demonstrated in the germ; and hydrolytic activity was found in the germ and endosperm.(1247).

Investigations showed that wheat and sorghum rapidly degrade dimethoate, *in vivo* and *in vitro*. While some oxidation of dimethoate did occur, initially the bulk of the material was degraded hydrolytically. On the basis of these studies, the following pathway was suggested (1248).

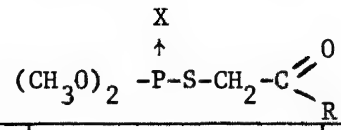


P = phosphatase O = Oxidase

C = carboxylase A = Amidase

After application of dimethoate to roots, leaves, or stems of bean plants (Phaseolus vulgaris L.), extraction and chromatography indicated the presence of eighteen metabolites. Of these, eight were identified: des-N-methyl dimethoate, dimethoate oxon analog, dimethoate carboxylic acid, des-0-methyl dimethoate, des-0-methyl carboxylic acid, 0,0-dimethyl phosphorodithioic acid, 0,0-dimethyl phosphorothioic acid, and dimethyl phosphoric acid (1732).

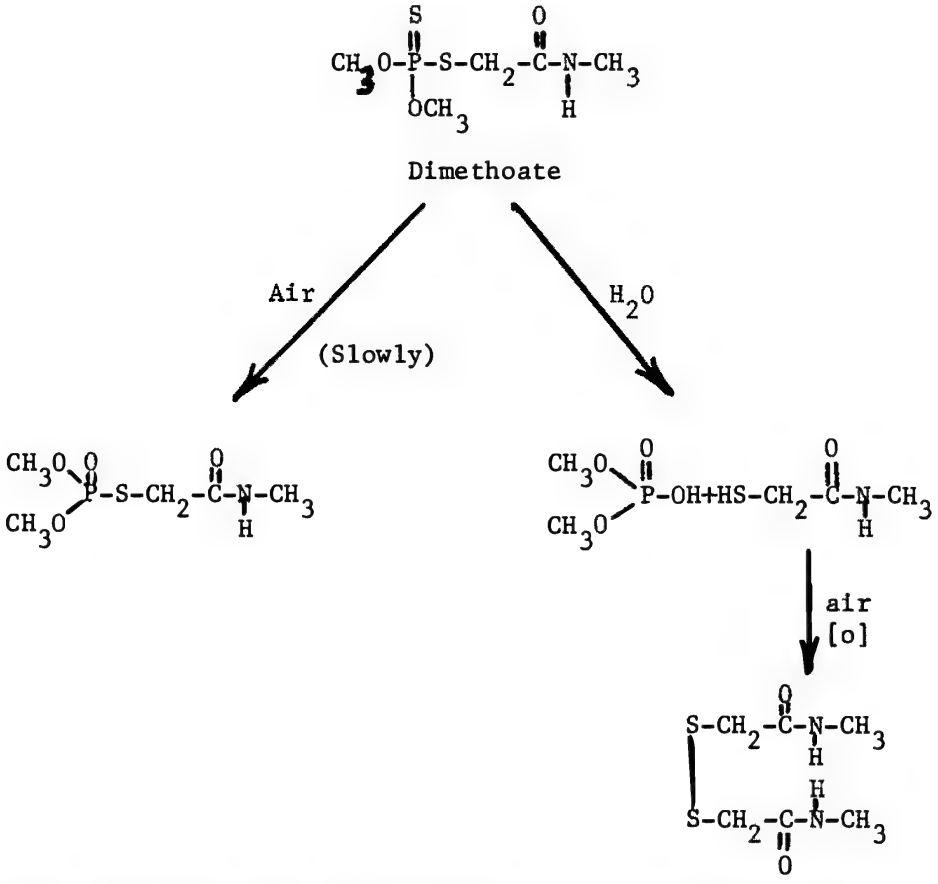
Tomato plants were treated with homologues of dimethoate, and their half lives were determined (1697, 1698).



	X	R	$t_{1/2}$(biol.)	$t_{1/2}$(pH=6@25°C)	$Q\left(\frac{t_{1/2}}{t_{1/2\ biol.}}\right)$
I	S	NH-CH_3	3.2	122	38.1
Ia	O	NH-CH_3	9.3	32.5	3.5
II	S	NH-C_4H_9(n)	2.5	168	67.2
III	S	NH-CH_2-$CH(CH_3)_2$	2.9	165	56.9
IV	S	NH-C-$(CH_3)_3$	1.8	208	114
IVa	O	NH-C-$(CH_3)_3$	6.7	105	15.7
V	S	-OH	--	--	--
Va	O	-OH	--	--	--

In sandy loam soil, dimethoate has half life of 2 1/2 days, where moderate rainfall occurs following application, to approximately 4 days during drought conditions (135).

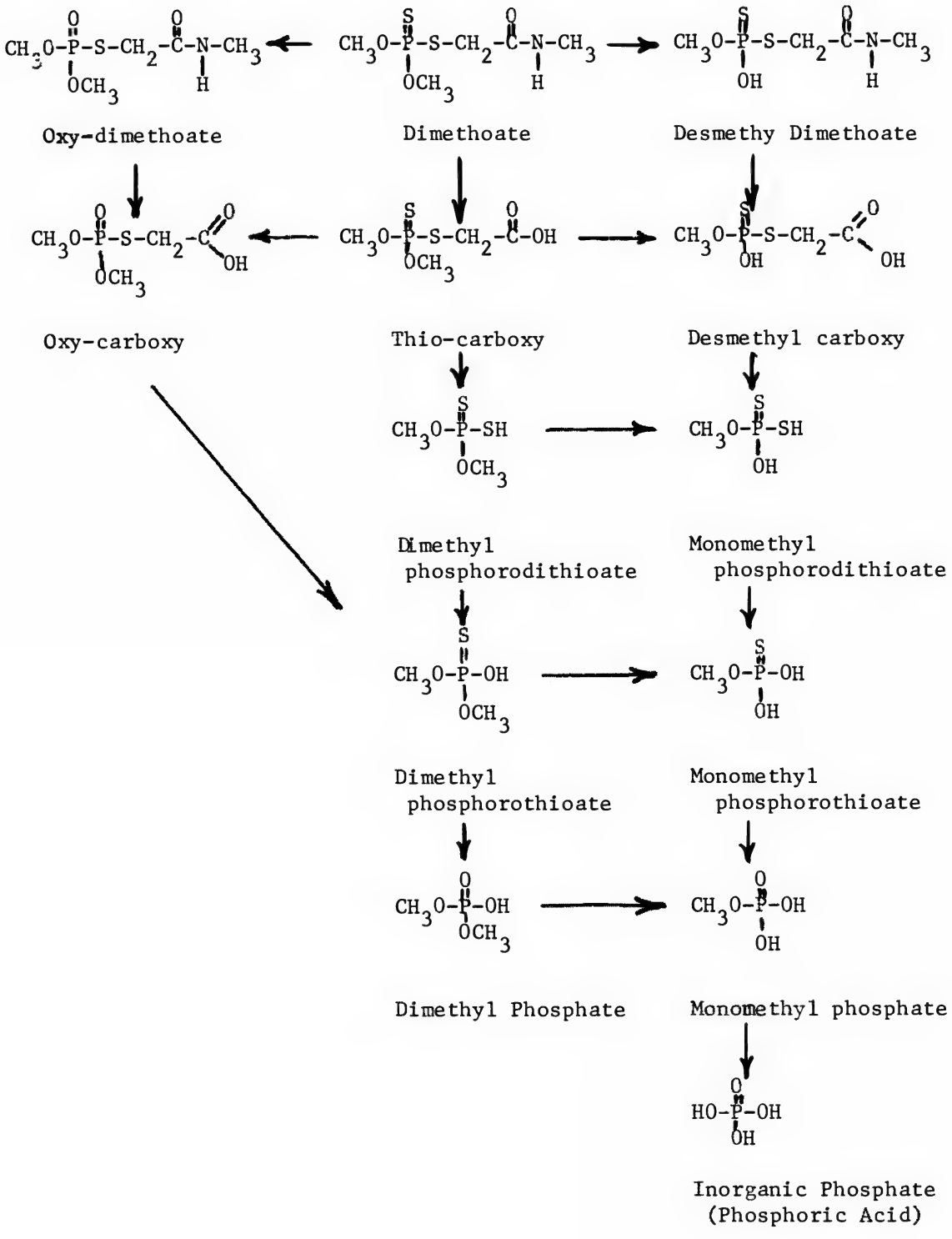
Dimethoate rapidly hydrolyzed in alkali (pH 11). In a half hour, approximately 50% of the dimethoate was degraded to water-soluble materials, predominantly desmethyl dimethoate (49.3%) 0,0-dimethylphosphoric acid (32.8%), the carboxy derivative (9.5%), and some 0,0-dimethyl phosphorodithioic acid (<0.1%) (167). In air the hydrolysis product thioacetamide was oxidized to the corresponding disulfide (1266).



When dimethoate was stored at 48 - 52°C in a pure state or in commercial concentrates, it lost potency because of hydrolysis, even in glassware. In galvanized containers the loss was apparent at 18 - 26°C. Hydrolysis affected the SCH_2 and PS bonds (1280).

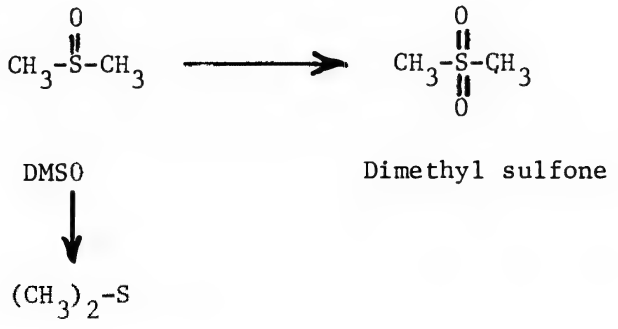
Dimethoate Homologs
Hydrolysis in Aqueous Buffered
Solutions (1697).

				Compound								
pH			°C	I	II	III	IV	Ia	IVa	V	Va
2	$t_{1/2}$	(d)	25	176	322	232	249	62.4	40.6	48.5(h)	0.46(h)
	k	(sec^{-1})		$4.56x10^{-8}$	$2.5x10^{-8}$	$3.46x10^{-8}$	$3.22x10^{-8}$	$1.29x10^{-7}$	$1.96x10^{-7}$	$3.9x10^{-6}$	$4.2x10^{-4}$
	$t_{1/2}$	(d)	40	26.4	41.2	32.8	36.4	11.9	8.1	10.7(h)	
	k	(sec^{-1})		$3.04x10^{-7}$	$1.95x10^{-7}$	$2.45x10^{-7}$	$2.21x10^{-7}$	$6.74x10^{-7}$	$9.90x10^{-7}$	$1.8x10^{-5}$	
	E_a	(kcal/Mol)		24.8	26.2	24.2	23.8	20.4	19.9	18.9	
4	$t_{1/2}$	(d)	25	160	240	212	249	73.8	113	14.2	8(h)
	k	(sec^{-1})		$5.01x10^{-8}$	$3.33x10^{-8}$	$3.77x10^{-8}$	$3.22x10^{-8}$	$1.09x10^{-7}$	$7.70x10^{-8}$	$5.56x10^{-7}$	$2.4x10^{-5}$
	$t_{1/2}$	(d)	40	29.1	36	32.4	36.4	14.5	20.2	2.9	
	k	(sec^{-1})		$2.76x10^{-7}$	$2.23x10^{-7}$	$2.47x10^{-7}$	$2.21x10^{-7}$	$5.54x10^{-7}$	$3.98x10^{-7}$	$2.76x10^{-6}$	
	E_a	(kcal/Mol)		21.1	23.5	23.3	23.8	20.05	21.2	19.6	
6	$t_{1/2}$	(d)	25	122	168	165	205	32.5	105	124	10.1
	k	(sec^{-1})		$6.59x10^{-8}$	$4.78x10^{-8}$	$4.86x10^{-8}$	$3.91x10^{-8}$	$2.47x10^{-7}$	$7.65x10^{-8}$	$6.47x10^{-8}$	$7.95x10^{-7}$
	$t_{1/2}$	(d)	40	22.8	30	28	29.9	6.9	15.25	13.5	
	k	(sec^{-1})		$3.53x10^{-7}$	$2.68x10^{-7}$	$2.87x10^{-7}$	$2.68x10^{-7}$	$1.16x10^{-6}$	$5.26x10^{-7}$	$5.95x10^{-7}$	
	E_a	(kcal/Mol)		20.8	21.4	21.9	23.8	19.1	23.8	27.4	
8	$t_{1/2}$	(d)	25	38.2	67.1	73.8	68.4	2.77	8.95	124	2.25
	k	(sec^{-1})		$2.1x10^{-7}$	$1.2x10^{-7}$	$1.09x10^{-7}$	$1.175x10^{-7}$	$2.9x10^{-6}$	$8.96x10^{-7}$	$6.47x10^{-8}$	$3.56x10^{-6}$
	$t_{1/2}$	(d)	40	6.46	9.46	10.75	10.4	13.5(h)	38.8(h)	13.8	
	k	(sec^{-1})		$1.24x10^{-6}$	$8.46x10^{-7}$	$7.46x10^{-7}$	$7.72x10^{-7}$	$1.43x10^{-5}$	$4.96x10^{-6}$	$5.81x10^{-7}$	
	E_a	(kcal/Mol)		22.0	24.2	23.8	23.2	19.7	21.2	27.1	
10	$t_{1/2}$	(h)	25	19.6	28.6	31.5	29.3	65.5(min)	2.47	19.6(d)	23.5
	k	(sec^{-1})		$9.82x10^{-6}$	$6.73x10^{-6}$	$6.11x10^{-6}$	$6.56x10^{-6}$	$1.76x10^{-4}$	$7.8x10^{-5}$	$4.1x10^{-7}$	$8.2x10^{-6}$
	$t_{1/2}$	(h)	40	2.12	4.2	5.52	5.8	15(min)	39.2(min)	2.8	
	k	(sec^{-1})		$9.08x10^{-5}$	$4.58x10^{-5}$	$3.49x10^{-5}$	$3.31x10^{-5}$	$7.7x10^{-4}$	$2.95x10^{-4}$	$2.87x10^{-6}$	
	E_a	(kcal/Mol)		27.4	23.8	21.6	20.0	18.25	16.4	24.0	


Oxy-dimethoate
Dimethoate
Desmethy Dimethoate
Oxy-carboxy
Thio-carboxy
Desmethyl carboxy
Dimethyl phosphorodithioate
Monomethyl phosphorodithioate
Dimethyl phosphorothioate
Monomethyl phosphorothioate
Dimethyl Phosphate
Monomethyl phosphate
Inorganic Phosphate (Phosphoric Acid)

Dimethyl Sulfoxide (DMSO)

The metabolic pathway for metabolism of DMSO was the same for all modes of exposure observed--oral, dermal or intraperitoneal. After exposure of rats, rabbits, guinea pigs and man to DMSO, the corresponding sulfone ($DMSO_2$) was observed in serum and/or urine (535, 697, 698, 701, 1505, 1661). Mixed melting point, elemental analysis and infrared were used to confirm the identity of the sulfone (697). In the exhaled breath of all animals tested, dimethyl sulfide was detected (821, 1505). The presence of this material in the breath of cats also was confirmed by means of gas chromatography and mass spectrography (372).



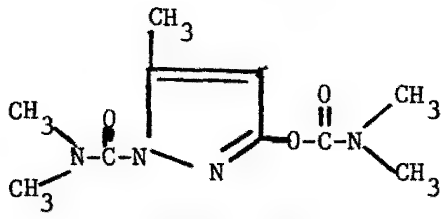
Dimetilan [2-Dimethylcarbamoyl-3-methyl-5-pyrazolyl dimethylcarbamate]

Labeled dimetilan was administered orally to rats. Within 4 hours after treatment, the urine contained 10% of the administered radioactivity and an additional 3% within the next 18 hours. Two metabolites were recovered but not identified (1545). Some production of CO_2 was observed (836).

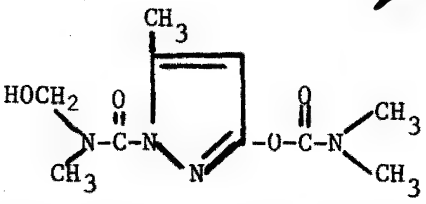
Other studies have shown a microsomal system, requiring TPNH and oxygen, that acts on N,N-dialkyl carbamates. Dealkylation occurs with formation of formaldehyde from N,N-dimethylcarbamates but not from N-methylcarbamates. Dimetilan was degraded by this system (676, 677); and 2-methylcarbamoyl-3-methyl-5-pyrazolyl dimethyl carbamate plus two unidentified compounds were observed (1111, 1112).

Dimetilan had a half-life in houseflies (Musca domestica L.), the American cockroach (Periplaneta americana L.), and the German cockroach (Blatella germanica L.) of less than 0.5 hours. Hydrolysis of the carbamate ester group accounted for some metabolism, since 13% of the injected dose was liberated as $C^{14}O_2$ within 1 hour after injection of dimetilan. Nine metabolites were detected from the German cockroach; 6 from the American cockroach; and 4 from the housefly. Chromatography indicated that the metabolite formed first and the major product was probably 2-dimethylcarbamoyl-3-methyl-5-pyrazolyl N-hydroxymethyl N-methylcarbamate. This could serve as a precursor for 2-(N-hydroxymethyl N-methyl)carbamoyl-3-methyl-5-pyrazolyl N-hydroxymethyl N-methylcarbamate, thought to be the structure of another of the metabolites found in both cockroaches but not in the housefly. A third metabolite found in all 3 insects co-chromatographed with 2-N-methylcarbamoyl-3-methyl-5-pyrazolyl dimethylcarbamate and exhibited the same biological activity (1544, 1545).

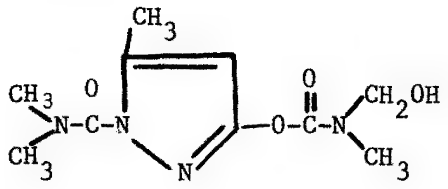
Exposure of dimetilan to ultraviolet radiation gave rise to three degradation products, one of which was identified as the monomethyl compound 3-methyl-5-hydroxy-pyrazol-5-dimethylcarbamate (391).



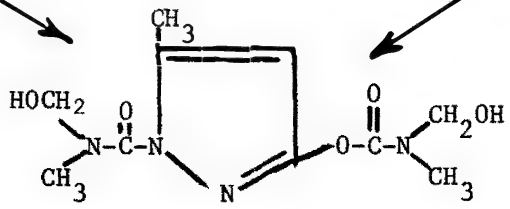
Dimetilan



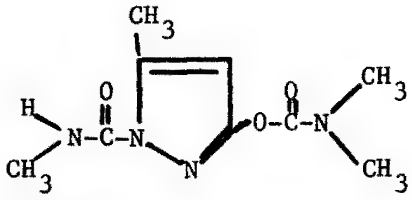
2-(N-hydroxymethyl, N-methyl) carbamoyl-3 methyl 5-pyrazolyl dimethylcarbmate



2-Dimethylcarbamoyl-3-methyl-5-pyrazolyl N-hydroxymethyl, N-methylcarbamate



2-(N-hydroxymethyl, N-methyl) carbamoyl-3-methyl-5-pyrazolyl N-hydroxymethyl, N-methylcarbamate



2-N-methylcarbamoyl-3-methyl-5-pyrazolyl dimethylcarbamate

Dinitro Compounds (Aromatic)

DNBP [2-sec-Butyl-4,6-dinitrophenol]

DNPP [2,4-Dinitro-6-isopropylphenol]

Binapacryl [2-sec-Butyl-4,6-dinitrophenyl 3,3-dimethylacrylate]

Paper electrophoresis of serum of rats poisoned by consumption, inhalation, or skin exposure to the herbicides dinitroisopropyl-(DNPP) and dinitrobutylphenol (DNBP) showed DNPP- and DNBP-albumin complexes. Liver, kidney, spleen, and blood contained metabolites. Although not identified, chemical reactions indicated that both herbicides were enzymatically reduced in the liver to primary amines (647, 648).

The urine of rabbits and rats fed DNBP and its esters were collected and examined for metabolites, Side chain oxidation and nitro reduction were observed. After feeding DNBP to rabbits, five metabolites were observed with paper chromatography. Similar results were obtained with DNBP-acetate and DNBP-1,1-dimethylacrylate. In addition to unchanged DNBP, 2-amino-4-nitro-6-sec-butylphenol and its glucuronide, β-methyl-β-(2-hydroxy-3,5-dinitrophenyl)propionic acid and an unidentified compound (416).

Binapacryl was absorbed poorly, if at all, through the skin of mice and rabbits. When administered orally to guinea pigs, this compound gives rise to DNBP in the blood. Death of experimental animals occurred only when the level of DNBP in the blood reached a critical level, indicating that toxic effects of binapacryl were caused by the DNBP produced in the tissues (144).

DNBP was degraded by Corynebacterium simplex but only negligible amounts of nitrite were observed (575).

Between 250 and 290°C, DNBP lost the OH group (1761).

Dinitro Compounds (Aromatic)

DNOC (4,6-Dinitro-o-Cresol;2,5-Dinitro-o-cresol)
[2,4-Dinitro-6-hydroxytoluene]

In man, DNOC acted as a cumulative poison and was eliminated slowly over a period of weeks. Small amounts were eliminated unchaged in the urine (1330).

When fed to rabbits, five percent was excreted unchanged and 1% as a conjugate. The main urinary metabolites were: 6-N-acetylamino-4-nitro-o-cresol (1 - 1.5%); O-conjugates of this compound (10%). 3-Amino-5-nitro-salicylic acid, 4-amino-6-nitro-o-cresol, and derivatives of this cresol were excreted in small amounts (1190, 1330, 1423).

After injection of DNOC in locusts Schistocerca gregaria and Locusta migratoria, 6-amino-4-nitro-o-cresol and its acetyl derivative were observed. When 6-amino-4-nitro-o-cresol was injected, the corresponding glucoside and sulfate were identified as well as the acetyl derivative (788).

Studies with soil microorganisms showed that aromatic dinitro compounds were slowly degraded (1308, 1663, 1664). When 2,4-dinitrophenol or 2,4-dinitro-o-cresol is exposed to bacteria (Pseudomonas) (1664) or an atypical Corynebacterium simplex (575) the yellow color bleached with the appearance of nitrite.

A Pseudomona (N.C.I.B. 9771) isolated from garden soil perfused with DNOC was capable of metabolizing this herbicide in pure culture. The degradative path, indicated by chemical isolation of intermediates, sequential induction, and other techniques, was determined (1397).

3,5-Dinitro-o-Toluamide

The metabolic fate of 3,5-dinitro-o-toluamide was investigated by feeding the carboxyl-C^{14}-labeled compound to chickens. Residues in tissues and fecal matter were isolated and identified by use of paper and column chromatography, infrared and visual spectroscopy, and X-ray analysis. In tissues, the parent compound and 3-amino-5-nitro-o-toluamide were recovered. The dinitro compound appeared to be "free" and was easily removed by simple acetone extraction. The amino compound was found and could be removed by acetone extraction only after enzymatic digestion. The metabolite accumulated preferentially in kidney and liver tissues while the parent compound was more or less uniformly distributed throughout the body. In fecal matter, 2-amino-5-nitro-o-toluamide and 3-amino-5-nitro-o-toluic acid were found (1323).

Dinitrophenol (DNP) [2,4-Dinitrophenol]

When DNP was administered subcutaneously to rabbits and rats, the compound was rapidly absorbed and distributed. Highest concentration was in serum. A large amount was also found in kidney, lung, and liver. In the urine of the treated rabbits, DNP-glucuronide, 2-aminonitrophenol and its ether sulfate were found (1057). In vitro, but not in vivo, studies indicated the presence of 4-aminoitrophenol. The latter was unstable; and it was assumed that, if this were formed in the body, the 4-ANP was further metabolized and could not be identified as such (1056).

DNP was reduced by rat liver homogenates to 4-amino-2-nitrophenol and some 2-amino-4-nitrophenol. Both were isolated from urine of rats administered 2,4-Dinitrophenol. Another compound observed was believed to be 2,4-diaminophenol but was not identified (1128).

Dinoben [2,5-Dichloro-3-nitrobenzoic acid]

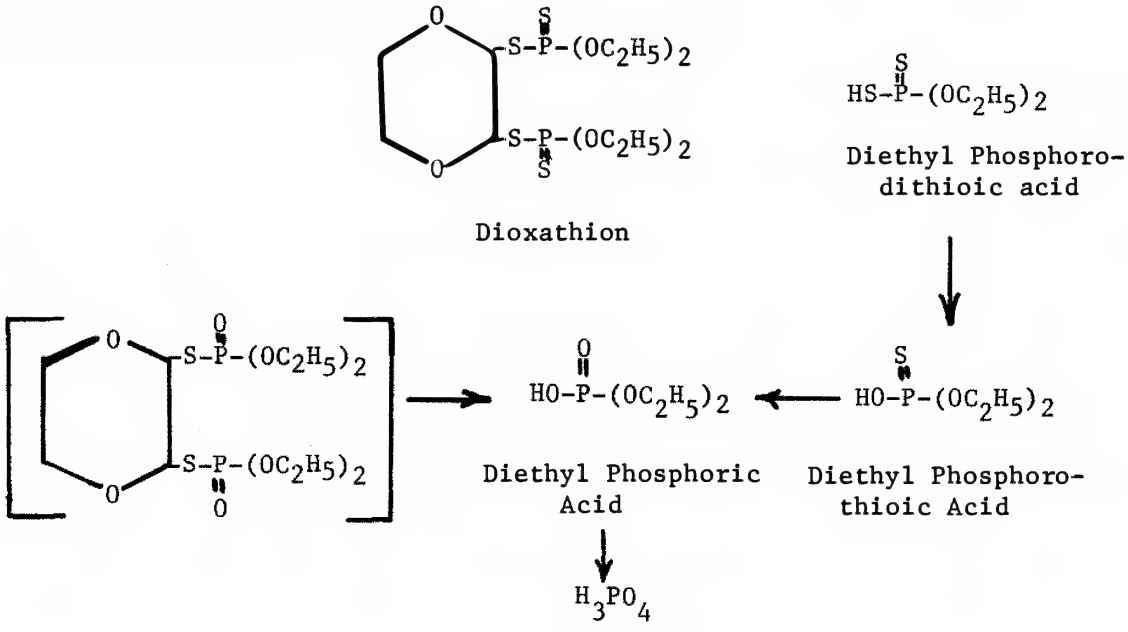
After treatment of soybean plants with dinoben-C^{14}, a conjugate was isolated that was chromatographically identical to the N-glycoside of amiben. Hydrolysis of this metabolite yielded a compound that was chromatographically similar to amiben-C^{14} and gave similar chemical reactions. It appeared, therefore, that soybean roots reduced the nitro group to an amine and conjugated the resultant compound with glucose (295). Dinoben-C^{14} hydroxypropyl ester was rapidly hydrolyzed by both corn and sudax to dinoben-C^{14}. This was reduced to amiben and converted to N-glycosides (296).

(See also Amiben.)

Dioxathion (Delnav)
[2,3-p-Dioxane S,S-bis(O,O-diethyl phosphorodithioate)]

When applied dermally to cattle, dioxathion was absorbed into the blood rapidly and the peak level of radioactivity was reached in 3 hours; whereas, in orally treated animals, it occurred in 12 hours. Blood radioactivity persisted at least one week after application. The radioactivity pattern of the urine followed that of the blood (264). Chromatography autoradiography showed that diethyl phosphoric, diethyl phosphorothioic, and diethyl phosphorodithioic acids were the major products of hydrolysis. Cis and trans dioxathion, dioxane derivative, and three unknown compounds were also found. A highly polar compound present at the origin point in the chromatography of the urine was presumed to be phosphoric acid (35). Analyses showed that dioxathion was present in the omental fat.

Dioxathion was hydrolyzed slowly on the plant surface but hydrolyzed rapidly when absorbed into the plant. Exposure to sunlight after application also caused formation of more polar derivatives and more potent cholinesterase compounds (238).



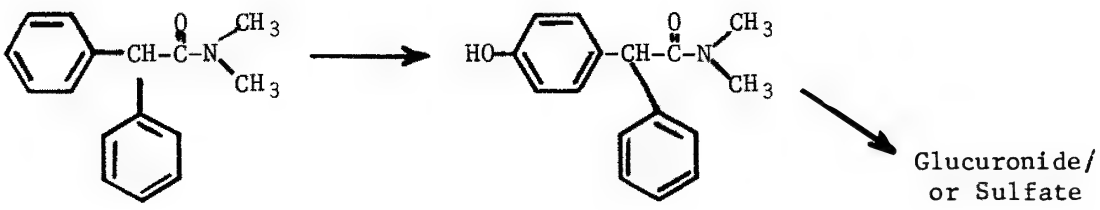
Diphenamid [N,N-Dimethyl-2,2-diphenylacetamide]

Labeled diphenamid was administered to male rats (Purdue-Wistar strain) orally, intraperitoneally, and subcutaneously. Half-lives were found to be 245, 235, and 210 minutes, respectively. Demethylation of diphenamid was found to be extensive; nordiphenamid was demethylated to a lesser extent. Analysis of urine showed the presence of nordiphenamid, diphenylacetamide, nordiphenamid N-glucuronide, N-methyl-N-hydroxymethyl diphenylacetamide O-glucuronide, p-hydroxydiphenamid, and p-hydroxy-nordiphenamid. The latter two were excreted as glucuronides and/or sulfates (961).

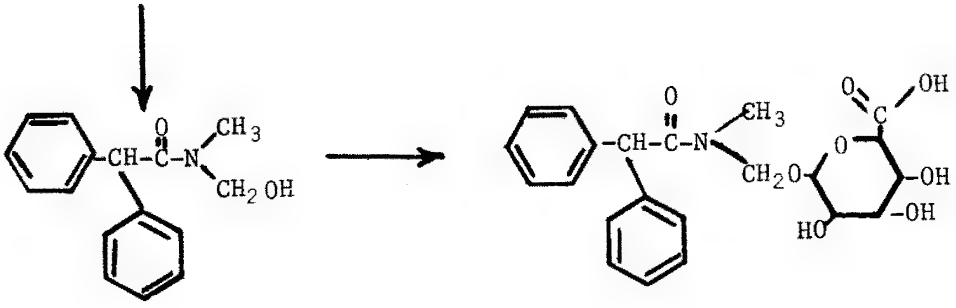
Strawberry plants were treated with labeled diphenamid. Extracts were examined by two T.L.C. systems and gas chromatography. Radioautographs of T.L.C. plates detected unchanged diphenamid and N-methyl-2-2-diphenylacetamide, diphenylacetamide, and diphenylacetic acid, p-hydroxy-diphenylacetic acid, α-hydroxy-diphenylacetic acid. Several additional compounds were implied but their identity were not established (555).

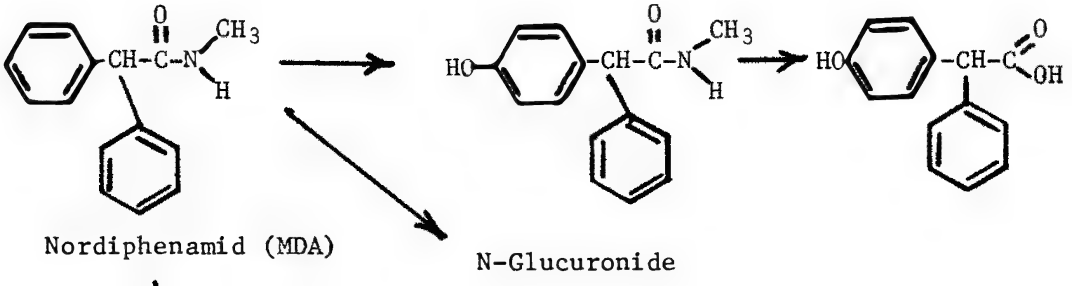
Studies were conducted with tomato plants. Diphenamid (59%); desmethyl diphenamid (36%); diphenylacetamide (5%); and some diphenyl-acetic acid were observed (862). Under sterile conditions, neither tomato plants nor barnyard grass metabolizes diphenamid within 16 days of application (length of experiment). Exposure of these treated plants to the soil fungi Trichoderma viride and Aspergillus candidus gave rise to both N-methyl-2,2-diphenylacetamide and 2,2-diphenylacetamide (783, 784). In soils, diphenamide gave rise to MDA, DA, the acid analog and some unknown compounds (554).

Aqueous solutions of diphenamid were irradiated with ultra-violet light at 2537A. Analysis of the resultant mixture showed the presence of benzoic acid, benzophenone, benzyhydrol, N-methyl-2,2-diphenylacetamide, and a compound believed to be N-formyl-2,2-diphenylacetamide. A polymeric material and some other unidentified compounds were also present (1236).



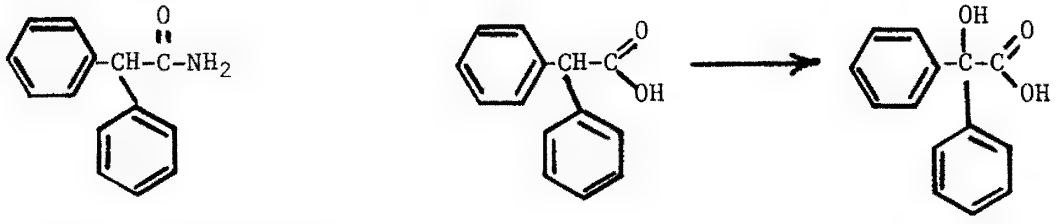
Diphenamid





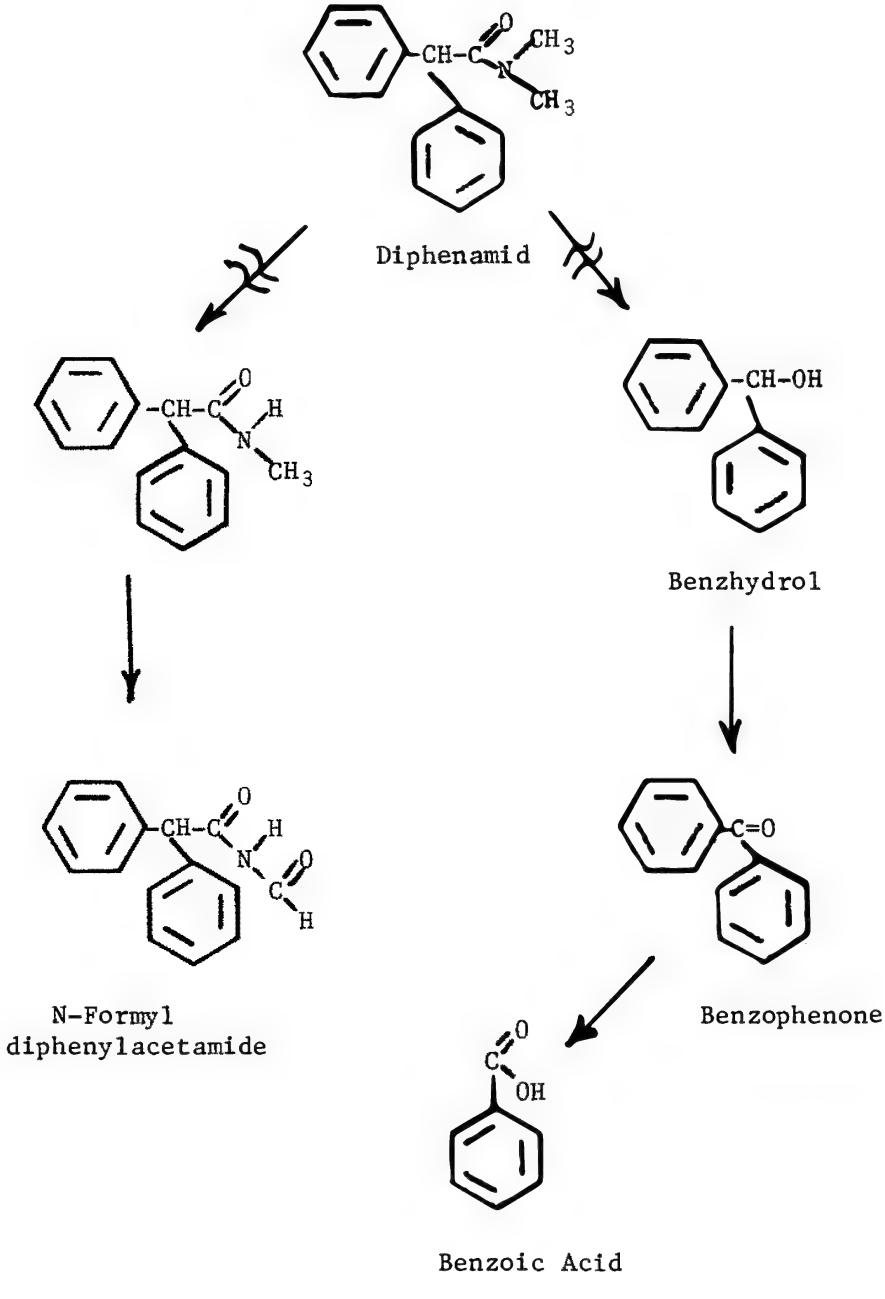
Nordiphenamid (MDA)

N-Glucuronide



Diphenylacetamide

UV-Irradiation



Diquat [1,1'-Ethylene-2,2'-dipyridinium dibromide]

Paraquat [1,1'-Dimethyl-4,4'-dipyridinium di(methyl sulfate)]

C^{14}-Diquat and paraquat, administered as a single oral dose to rats, appeared in feces within 2 days. The percentage of the dose appearing in the urine seemed to depend on the strain of rat used. After an oral dose, the bulk of the material appeared in the feces; whereas after a subcutaneous dose, most of the diquat and paraquat was in the urine. There was no marked biliary excretion. Comparison of colorimetric and radioactivity determinations on feces indicated that about 70% of the oral dose of diquat and about 30% of an oral dose of paraquat appeared in feces in a degraded form. Incubation with a fecal homogenate, heat treated and not heat treated, demonstrated that the degradation was the result of microbiological action (336).

After administration of labeled diquat and paraquat to dairy cattle, only about 0.001 - 0.015% of the ingested dose appeared in the milk. Analyses indicated several metabolites arose from diquat. Very little (less than 3%) of either compound appeared in the urine. Most of the diquat ingested was excreted on the second day after dosage; and, after eleven days, no measurable radioactivity was present. Tissue levels were very low except in organs of excretion (1361).

After feeding dairy cattle and sheep on diquat-treated silage, no residues were detected in the milk of the cattle nor in the meat or organs of the sacrificed animal. Incubation of diquat with sheep feces or rumen liquor resulted in an appreciable loss of diquat (119).

Studies with labeled material indicated that those compounds were not degraded by alligatorweed. Both compounds reacted as free radicals (510, 686).

Paraquat dihydrochloride was applied to maize, tomato, and broad bean plants as droplets of an aqueous solution. There was little or no loss of paraquat on plants kept in darkness. When treated plants were exposed to sunlight, 4-carboxy-1-methylpyridinium chloride and methylamine were detected (1319).

Isolation of micro-organisms were made from a wide variety of soils, some of which had been treated with paraquat prior to sampling. Corynebacterium fascians (Tilford) Dowson, Clostridium pasteurianum Winogradsky and some unidentified anerobes, and Lipomyces starkeyi, a yeast, were able to degrade paraquat (76). Reduction of paraquat and diquat to colored free radicals was observed in fungal and bacterial cultures. In some cases, the organisms were colored, indicating entrance

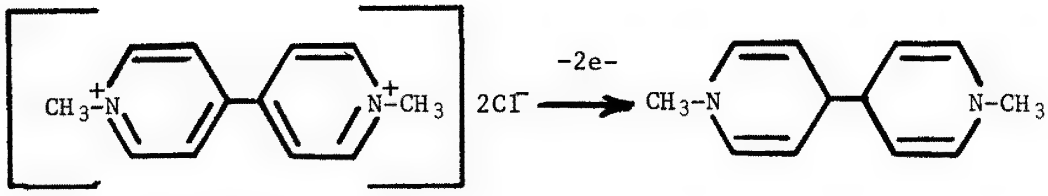
into the organism or adsorption of the herbicide to cell walls. The fungus, Cephalosporium sp., showed little or no degradation of paraquat whereas an unidentified bacterium produced at least two degradation products (156).

After seven days exposure to ultraviolet light, 85% of the radioactivity of C^{14}-labeled diquat and paraquat was lost. Electrophoresis indicated three degradation products from paraquat and one or more from diquat (287). After irradiation of paraquat in aqueous solution and in the presence of oxygen, the NMR spectrum of one of the products was consistent with a methyl quaternary pyridinium compound substituted at the 4-position. Elemental analyses, paper chromatography, IR and UV analyses showed that the compound was the N-methyl betaine of isonicotinic acid. Further irradiation of the latter gave rise, via carbonyl compounds, to methyl amine and 4-carboxy-1-methylpyridinium chloride. It was also shown that this reaction occurred on sprayed plants and that maximum breakdown of paraquat occurred when the chemical remained largely on the surface of treated leaves in sunlight (220, 508, 1316, 1317, 1318, 1632).

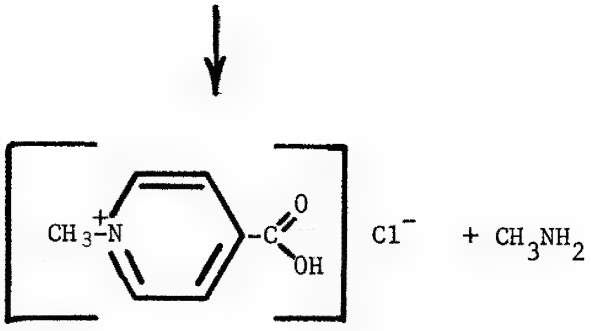
In the absence of oxygen, UV irradiation of parquat gave rise to a brown resin, presumably polymeric (1317).

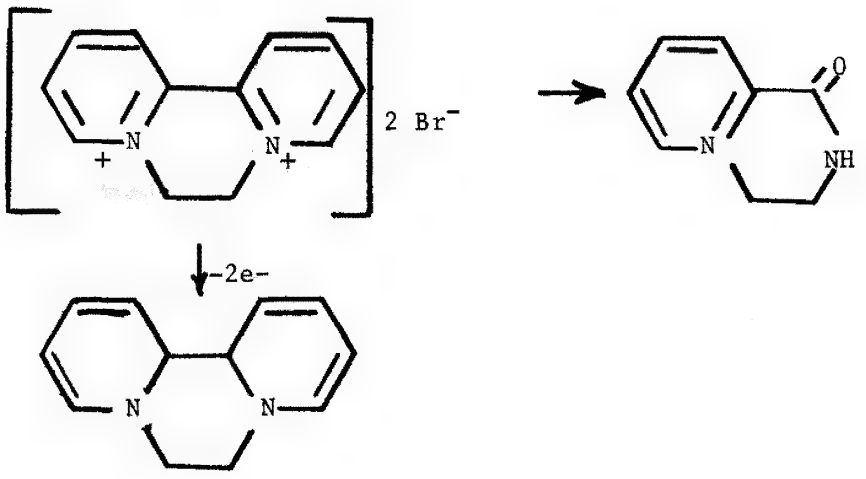
Diquat underwent photochemical degradation in solution or after application to plants. Using C^{14}-ring- or -ethylene bridge-labeled diquat, one major metabolite and several minor compounds were observed by paper chromatography. That the major metabolite was radioactive in both instances indicated at least one intact ring. Chemical reaction indicated the presence of nitrogen; an elemental analysis, together with infra-red, ultraviolet, nuclear magnetic resonance, and mass spectra showed the compound to be tetrahydro-oxo-pyrido-pyrazine. Structure was confirmed by synthesis and comparison of infra-red spectra and chromatographic properties (1321).

Studies indicated that diquat and paraquat were strongly adsorbed, in similar amounts, to mineral soils kaolinite and montmorillonite; however, only montmorillonite appeared to hold herbicides in an unavailable form to wheat at extremely high rates. When added to plastic pools, montmorillonite drastically reduced paraquat solution concentration within 24 hours (287, 811, 1429). Addition of montomorillonite clay to solutions of diquat, in an amount calculated to adsorb one half of the C^{14}-diquat, reduced $C^{14}O_2$ evolution to approximately one half. When sufficient montmorillonite was added to adsorb all of the diquat, no $C^{14}O_2$ was detected (1598).



Paraquat





Disulfoton (Di-Syston) [0,0-Diethyl S-(2-ethylthioethyl) phosphorodithioate]

After intraperitoneal injection of labeled di-syston into rats, the rats were sacrificed and the livers analyzed. Di-syston sulfoxide, di-syston sulfone, di-syston-P=0 sulfoxide and di-syston-P=0 sulfone were found. In the urine collected from treated rats, only hydrolytic products were identified: diethyl phosphate, diethyl phosphorothioate, diethyl phosphorodithioate, and inorganic phosphate. Four other compounds were observed but not identified. The di-syston phosphorothiolate derivate was not found (197).

Di-syston-P^{32} was injected into bollworm larvae which were sacrificed at varying intervals. Di-syston sulfoxide and sulfone, di-syston-P=0 sulfoxide and sulfone, inorganic phosphate, ethyl phosphate, diethyl phosphate, diethyl phosphorothiolate and several unidentified compounds were found. Examination of the excreta gave similar results. After exposure of tobacco budworms to labeled di-syston, the same products were found. However, the rate of metabolism of di-syston and its metabolites was greater (197).

In vegetables, di-syston was first oxidized to P=0. Thereafter, the thio group was converted successively to -SO- and $-SO_2-$ (1107).

Cotton leaves and seedlings were treated with labeled di-syston. Initial oxidation of the 2-ethylthio ether was very rapid. After one day, di-syston sulfone was the major metabolite. After 16 days, the major metabolite was di-syston-P=0 sulfone. The only hydrolytic products found were inorganic phosphate, diethyl phosphate, and diethyl phosphorothioate (197). Similar results were obtained with lemon, bean, and alfalfa plants (992). Metabolites of di-syston on onions were identified as the P=0 analog, the sulfone and sulfoxide (1107).

Adsorption of disulfoton by soil was studied by a wet slurry technique. Extraction studies of soils and solutions after equilibration showed that some disulfoton was lost by microbial alteration and adsorption by glass. The empirical Freundlich isotherm fitted adsorption results for 17 different soils. The regression of log b on the log of the organic carbon content of the soils was highly significant. The fit was further improved by including pH as a second dependent variant. This probably reflected differences in the nature of the organic matter formed in soils of different acidity (562).

Storage stability studies of di-syston on fertilizers indicated good stability at room temperature for periods up to one year with recoveries of 70-90%. Exceptions to this were observed with ammonium nitrate and superphosphate; only traces of di-syston were detected. The main oxidation products were the oxygen analogs of the sulfoxide and sulfone (705).

Thin films of di-syston were irradiated with U.V. light with a wavelength maximum of 2540A. Three compounds were observed. It was believed that disulfoton sulfone and sulfoxide were present. The third compound was not characterized but may have been an oxygen analog (1608).

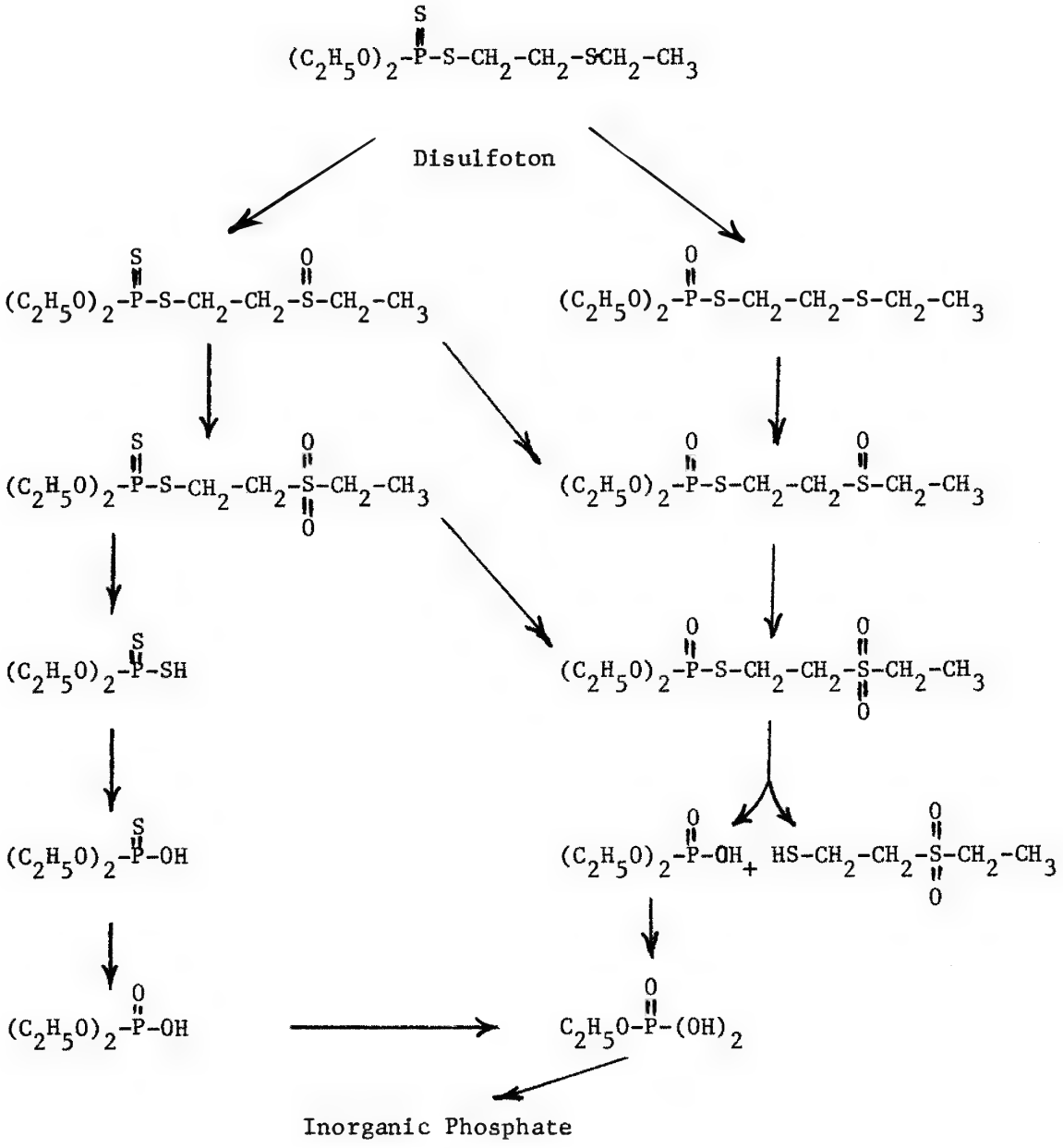
Kinetic studies have shown that temperature, by its general effect on chemical kinetics and by governing the rate of physiological activities of the plant, plays a large but somewhat predictable role in the metabolism of systemic insecticides in plants. Figures showed that for every rise of 10°C, the rate of disappearance of di-syston sulfoxide increased by about 1.86 times. Similar effects are presumed for the reactions giving rise to the other identified metabolites (1002).

Oxidation of di-syston by aqueous bromine, ascorbic acid - Fe++, or horseradish peroxidase gave $(C_2H_3O)_2P(O)SCH_2CH_2SC_2H_5$, the sulfoxide and the sulfone in all cases (1107).

Uptake by different soils (562)

	Soil	b	K	n	significance †
1.	Broadbalk FYM plot	21·6	21·5	1·00	
2.	Broadbalk nil plot	5·9	5·8	1·01	
3.	Woburn plot 2	20·0	20·0	1·00	
4.	Woburn plot 4	17·0	20·5	0·85	*
5.	Woburn plot 34	13·3	14·9	0·93	
6.	Stretham	10·2	14·7	0·81	**
7.	Isleham 1	43·2	49·1	0·87	**
8.	Isleham 2	32·4	55·5	0·88	**
9.	Bottisham	57·5	58·6	0·97	
10.	Worlington	6·2	5·3	1·09	
11.	Spinney	85·5	95·7	0·8	**
12.	Wicken	15·9	20·3	0·8	**
13.	Prickwillow	100·7	100·5	1·00	
14.	Moulton	15·8	21·1	0·81	**
15.	Oakington	14·8	16·0	0·92	
16.	Peacock	68·6	70·3	0·94	
17.	Adventurers	193·6	134·6	0·95	

† The symbols * and ** indicate that the Freundlich equation gives a fit significantly better than the straight line at 5% and 1% levels respectively


Disulfoton
Inorganic Phosphate

Dithiocarbamates

Dimethyldithiocarbamate
Ethylene bis(dithiocarbamate)
Methyldithiocarbamate

Dimethyldithiocarbamate (DDC)

Ferric Salt (Ferbam)
Sodium Salt (NaDDC)
Zinc Salt (Ziram)

(See also TMTD and TTD)

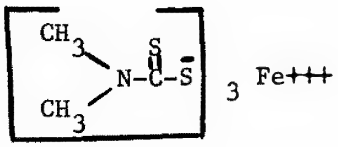
NaDDC

After uptake of the fungicide sodium dimethyldithiocarbamate by the roots of cucumber plants, three other fungitoxic agents could be distinguished on paper chromatograms. Studies with bean plants gave similar results. Incubation of potato chips in a solution of dimethyldithiocarbamate produced two of the compounds. One of these was identified by ultraviolet, infra-red, and optical rotation as dimethyldithiocarbamate-β-glucoside. By similar methods, another compound was isolated and identified as β-(dimethylthiocarbamoylthio)alanine. A third compound was identified as optically active thiazolidine-2-thione-4-carboxylic acid. This latter probably arises through non-enzymatic decomposition of the alanine derivative (361, 362, 363, 364, 365, 766, 761, 1301).

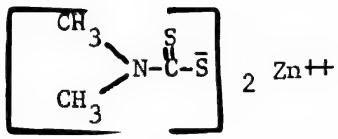
From suspensions of yeast cells (Saccharomyces cerevisiae) and sodium dimethyldithiocarbamate, a compound identified as γ-(dimethylthiocarbamoylthio)-α-aminobutyric acid was isolated. Another unstable compound, thought to be the corresponding keto acid, was found but not completely identified. Similar results were obtained with many other soil microorganisms. A scheme involving Cysteine and homoserine was proposed (1301, 1304).

DDC, labeled with C^{14} in the methyl groups and S^{35} in the dithiocarbamate, was applied to leaves and roots of papaya seedling (Carica Papaya) as the potassium or zinc salt. Part of the fungicide was hydrolyzed to CS_2 and dimethylamine. An appreciable amount of S^{35} appeared in the sulfur amino acids, free and in protein, and an inorganic sulfate. $C^{14}O_2$ was collected and some C^{14} was found in many plant constituents. The largest amount of foliarly applied radioactivity was recovered from cell sap as dimethyldithiocarbamyl glucoside. Lesser amounts were present as dimethyldithiocarbamyl alanine and four other compounds not identified (1557).

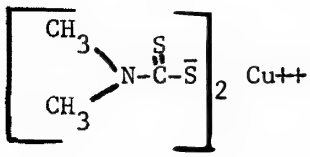
Ferbam



Ziram

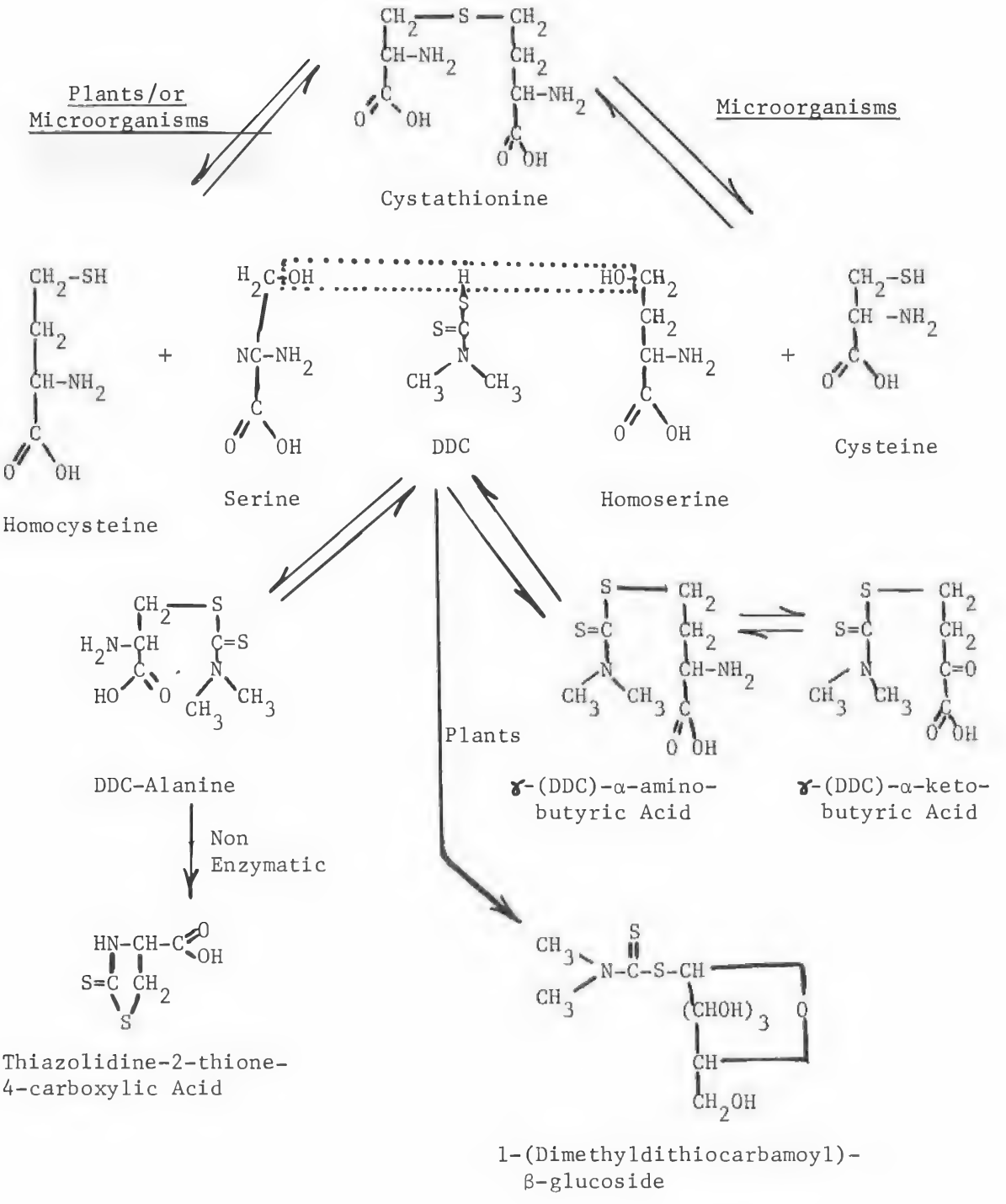


Copper Salt



Ferbam reacts with thiols by a radical exchange mechanism. The final products are the corresponding disulfide of the parent thiol and dimethyldithiocarbamate. At pH >7, ferbam decomposes to give dithiocarbamate ions. At pH <5, it decomposes to dimethylamine and CS_2. At pH 6 to 7, the nature of the buffer is important: ferbam is stable in acetate but decomposes in phosphate buffer to dithiocarbamate (1123).

At acid pH, ultraviolet spectra showed that $[(CH_3)_2NCS_2]_2$ was present. At higher pH, the spectra indicated the presence of $(CH_3)_2$-N-CS_2^-. In the presence of dilute acid, ferbam and ziram decomposed at a slower rate than did the sodium salt, with evolution of carbon disulfide. A secondary decomposition product in the form of an amine also formed. When the copper salt was tested, no carbon disulfide could be detected (897, 1407, 1474).


Plants/or
Microorganisms
Microorganisms
Cystathionine
Homocysteine
Serine
DDC
Homoserine
Cysteine
DDC-Alanine
Non
Enzymatic
Plants
γ-(DDC)-α-amino-
butyric Acid
γ-(DDC)-α-keto-
butyric Acid
Thiazolidine-2-thione-
4-carboxylic Acid
1-(Dimethyldithiocarbamoyl)-
β-glucoside

Ethylene bis(dithiocarbamate)

Nabam = Disodium salt
Maneb = Manganese salt
Zineb = Zinc salt

The effectiveness of ethylene disdithiocarbamates was thought to be dependent on the formation of ethylene thiuram monosulfide and its ultimate conversion to isothiocyanate which may react with sulfhydryls. The pH conditions, type and amount of inorganic salt present, and the order of mixing, all affected these conversions. Some free ethylene bisdithiocarbamic acid was always present in solutions of its salts because of hydrolysis. Salts of the acid degraded to ethylene thiuram monsulfide and polymeric ethylene thiuram monosulfides (88, 310, 805, 1034, 1129, 1302, 1305, 1310, 1442).

Except for Cu++ salts, dithiocarbamates inhibited polyphenol oxidase by complexing the enzyme or increasing metal dissociation. Inductance effects of the amine "N" substituents and the tautomerism of the "S" atom to sulfhydryl played an important role in this aspect. Phosphorylation of glucose and mannose was interrupted through inhibition of 6-phosphogluconate and glucose-6-phosphate dehydrogenase (384, 739, 906, 907, 1118, 1119, 1306, 1310).

Nabam

Above aqueous Nabam solutions or treated soil, in addition to H_2S, CS_2 and ethylenediamine, carbonyl sulfide was also observed and identified by means of gas chromatography and infra-red (1023, 1053). In the presence of an acid buffer, H_2S and CS_2 were evolved. In distilled water, nabam underwent hydrolysis with evolution of carbon disulfide. The latter underwent oxidation to sulfate. No hydrogen sulfide was evolved (897). Traces of manganese catalyzed the oxidation of nabam to ethylenethiuram monosulfide (905).

Maneb

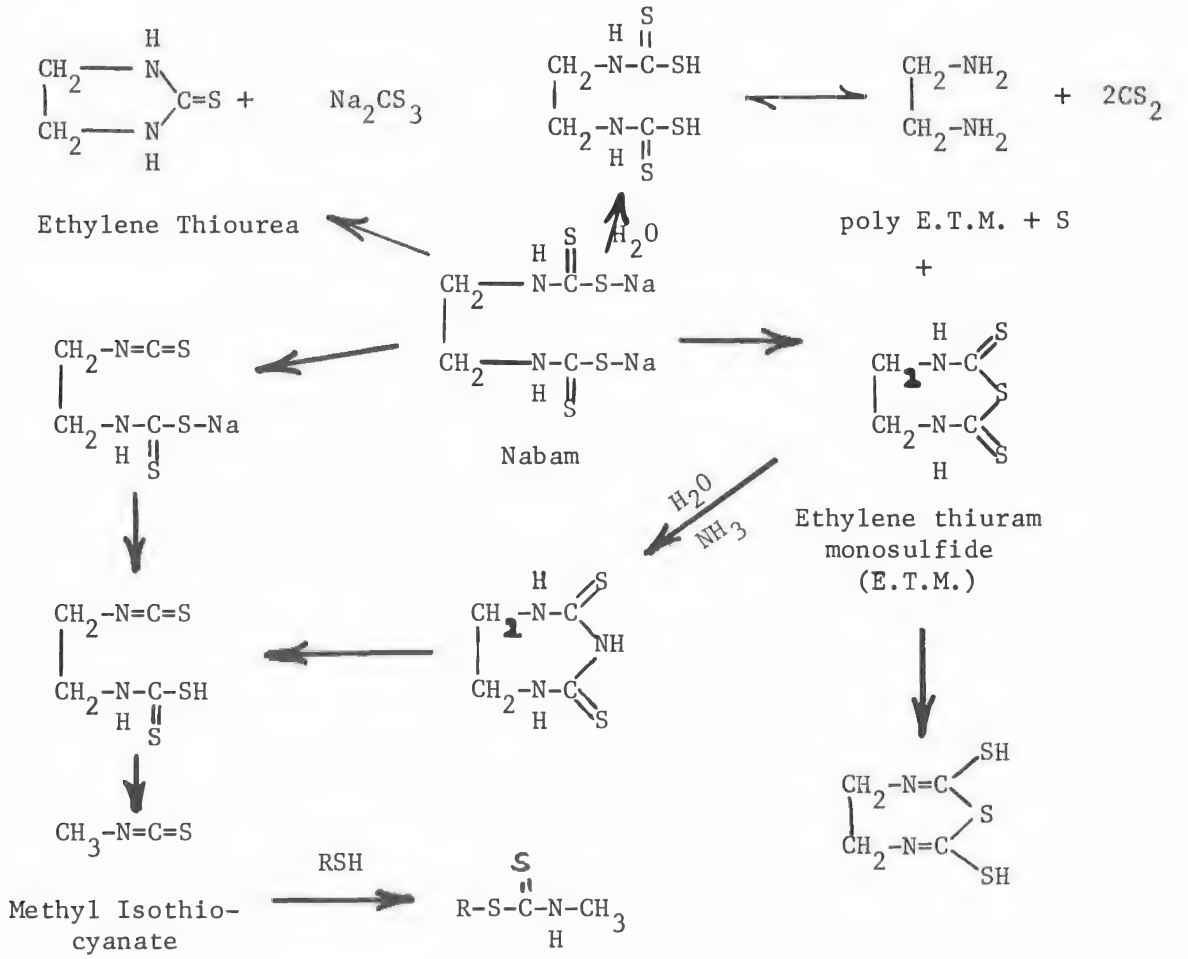
Aqueous suspensions of maneb decomposed rapidly. At ordinary temperatures, 10% of the maneb decomposed in one hour; at 40°C, decomposition was almost complete in 3 hours. In the absence of oxygen, CS_2, H_2S, and ethylenethiourea formed. In the presence of oxygen, carbon disulfide (but not hydrogen sulfide) was liberated. A portion of maneb was insoluble in water. This was partly soluble in $CHCl_3$. Paper chromatography indicated four compounds, one of which was identified by infrared as ethylenethiourea. Two other compounds were tentatively identified as ethylenethiuram mono- and disulfides (408, 1447). Sulfate was also characterized.

When stored in the dark maneb underwent decomposition to ethylenethiourea, ethylenethiuram mono- and di-sulfide, ethylenediamine, sulfur and several unidentified compounds (326).

Zineb

Zineb underwent decomposition, giving rise to ethylenethiuram monosulfide, ethylenethiourea, carbon disulfide and zinc sulfide (408, 1157). After storage of zineb in darkness, ethylenethiourea, ethylenethiuram mono- and disulfide, a trace of sulfur and one unidentified compound were observed (326).

The zinc salt showed no degradation when exposed to sunlight for varying period up to 1 1/2 hours. In soil, degradation of zineb occurred but was not complete even after 45 days (709).

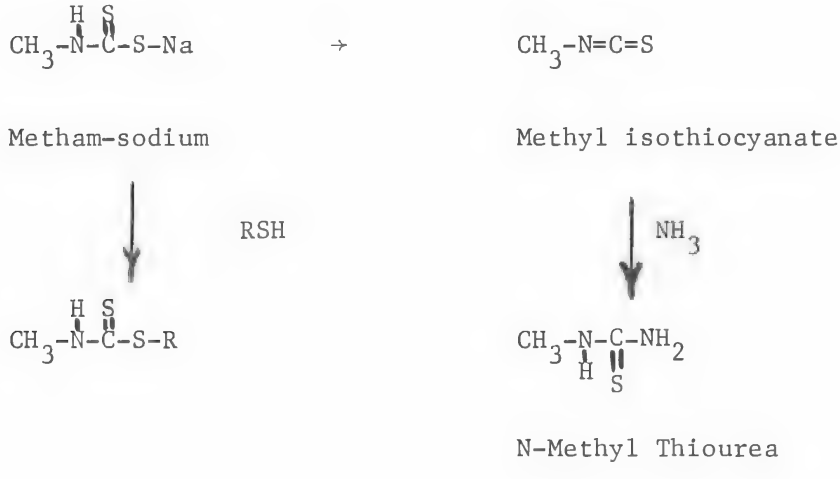


Methyldithiocarbamate

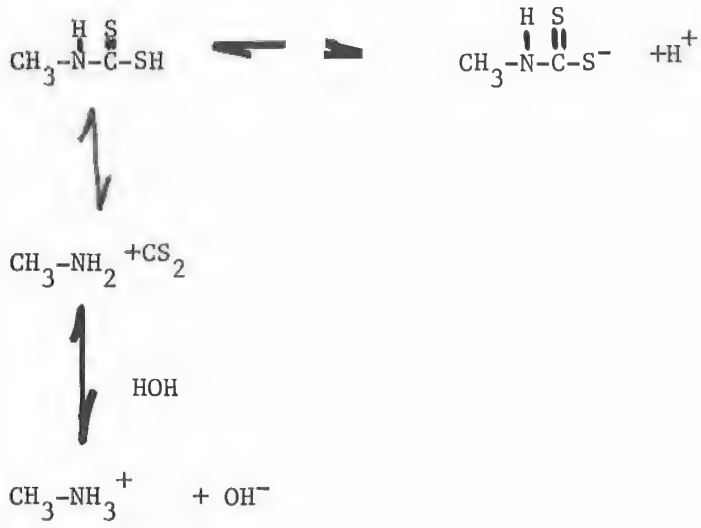
Metham Sodium (Vapam) [Sodium N-methyldithiocarbamate]

The principal product of decomposition of metham-sodium, when applied to soil, has been shown to be methyl isothiocyanate. This can then react with sulfhydryls or with ammonia to form N-methylthiourea. However, under outdoor conditions, this latter reaction is not significant (36, 37, 279, 702, 890, 1310).

In dilute aqueous solutions, at pH 9.5, vapam decomposes to methyl isothiocyanate and sulfur. Under acid conditions, carbon disulfide, hydrogen sulfide, N,N'-dimethylthiuram disulfide, methylamine, and methyl isothiocyanate formed. Methylamine and carbon disulfide can react to form methyl isothiocyanate. This may then react with vapam to produce dimethyl-thiuram disulfide and with methylamine or hydrogen sulfide to form dimethylthiourea.(1432).



In a closed aqueous system, metham-sodium gave rise to methyl isothiocyanate and hydrogen sulfate. Apparently these interacted and a white precipitate formed. The latter exhibited an ultraviolet spectrum like that of bis (N-methylthiocarbamyl) disulfide. The pH of the water was 7.5 and was probably buffered by a three-way equilibrium:



Esterification of metham-sodium then apparently occurred by direct addition of the halogenated hydrocarbon (1010).

Dodecylguanidine (Dodine, Cyprex) (N-Dodecygluanidine acetate)

Studies have shown that dodecylguanidine inhibits oxidation of glucose by cells of the fungus (Saccharomyces pastorianus). Other studies attribute the toxicity of dodine to the blocking of vital anionic sites at the cell surface and inactivation of enzymes. No metabolites have been demonstrated (192).

Some degradation of C^{14}-dodine occurred after application to apple trees. Traces of radiotagged carbon appeared in the fruit. Apparently, most of the radioactivity settled in the protein or peptide portion of the fruit. One of four compounds detected appeared to be guanidine or guanidine monosubstituted with a small group (324).

Dyrene (Kemate) [2,4-Dichloro-6-anilino-s-triazine]

It has been shown that through a metathetic reaction involving the ring chlorine, this compound can combine with metabolic intermediates containing amino and sulfhydryl groups (205).

Dursban [3,5,6-Trichloro-2-pyridyl phosphorothioate]

Dursban was stable when incubated with fresh rumen fluid or beef liver. When fed to cows, some unchanged dursban was found in feces but not in the urine or milk. Diethylthiophosphate and diethylphosphate were excreted in the urine (590). The oxon occurred in sprayed cattle (1704).

After single doses of Cl^{36}-dursban were fed to rats, 90% of the radioactivity rapidly appeared in the urine and 10% in feces. Compounds IV and V were identified. However, only dursban itself seemed to accumulate in the tissues and this was essentially in the fat (1327).

The metabolism of dursban in fish was studied in a tank containing soil and plants. After exposure to dursban, the fish were sacrificed and the fish and some water examined via paper chromatography. In addition to the oxygen analog (II) of dursban, the monoethyl analog (III) of dursban and its oxygen (IV) analog, 3,5,6-trichloro-2-pyridyl phosphate (V), and 3,5,6-trichloro-2-pyridinol (VI) were also found. In the fish tissues themselves, compounds II, IV, V, VI were found (1325).

Cranberry beans were grown in a nutrient solution containing 50 ppm Cl^{36} Dursban. Some dursban was apparently lost as a result of the continuous aeration of the nutrient solution. Chromatography of plant extracts indicated the presence of seven metabolites. Of these, four were identified as: 3,5,6-trichloro-2-pyridyl phosphate; 3,5,6-trichloro-2-pyridinol; Ethyl 3,5,6-trichloro-2-pyridyl phosphate; O-ethyl-O-3,5,6-trichloro-2-pyridyl phosphorothioate (1328).

C^{14}-and Cl^{36}-labeled dursban was applied to cranberry bean and corn leaves. Within three days, about 80% of the radioactivity was lost, presumably by volatilization. The remainder was slowly metabolized. Cl^{36}-chloride was found in the plants, indicating dehalogenation of the pyridinol had taken place. Analyses also showed the presence of 3,5,6-trichloro-2-pyridinol. The low level of radioactivity present in the plants made it impossible to obtain definite proof that compounds III and IV were present as plant metabolites or UV breakdown products (1326).

Dursban was very stable under neutral or slightly acid conditions at room temperatures. Breakdown increased with pH. The half-life in water (pH not given) or soil was about 80-100 days. Exposure of crystalline dursban to UV converted it slowly from white to a brown color. In solution, decomposition was more rapid and complete. Chlorine was replaced by hydroxyl groups which could undergo oxidation to keto groups.

These diones or triones could be oxidized, forming a series of colored derivatives. These polyhydroxyl compounds were oxidized with ring cleavage. After prolonged irradiation only CO_2,$(NH_4)_2CO_3$, and NaCl, and traces of HCOOH and an amine compound were found (1324, 1624).

Variability of the rate constant at various time intervals indicated that hydrolysis other than that which produced 3,5,6-Cl_3-2-pyridinol must occur also. Possibly nucleophilic displacement of the ethoxy group may be involved (194).

pH	K(Days^{-1})	Estimated Half-life Days
3.97	1.98×10^{-3}	350
4.95	6.43×10^{-3}	1080
6.00	3.6×10^{-4}	1930
Methanol	2.05×10^{-3}	340
8.00	1.39×10^{-2}	50
8.95	2.39×10^{-2}	29
9.96	9.50×10^{-2}	7.2
12.00	3.66	0.19

Dyfonate [O-Ethyl S-phenylethyl phosphonodithioate]

The metabolism of dyfonate in rats was studied by means of labeled insecticide. When C^{14}-ethoxy labeled dyfonate was administered, 95% of the label was found in the urine; 5% in feces; and only a trace (0.5%) in exhaled air. With S^{35}-label, 63% of the dose was recovered in urine; 32% in feces; 2% in tissues; and 0.1% in exhaled air. Diethyl phosphate and thiophosphate, thiophenol, methylphenylthioether, and thiolate analog of dyfonate were observed (981).

Endosulfan (Thiodan) [6,7,8,9,10,10-Hexachloro-1,5,5a,6,9,9a-hexahydro 6,9-methano-2,4,3-benzodioxathiepin-3-oxide]

When endosulfan was administered orally or intraperitoneally to rats, both isomers were excreted in part unchanged. Examination of gall, feces and urine and re-feeding of metabolites was used to study the metabolic path. In addition to the sulfate, endosulfan hydroxyether and the corresponding lactone were found after application of endosulfan. Administration of the sulfate gave rise to the hydroxyether and lactone. The hydroxyether and the lactone were observed after administration of either compound. When endosulfan ether or endosulfandiol was administered to rats, in addition to these two materials, the hydroxyether and lactone and one unidentified compound were found (879, 1395, 1593). Studies with rabbits and guinea pigs indicated metabolites were excreted via urine as conjugates but none were identified (1191).

In other studies, white mice (BALB/c strain) were fed C^{14}-labeled endosulfan. The two isomers were not completely absorbed from the gastrointestinal tract but, along with endosulfan sulfate and diol, were excreted in the feces. Only a trace of oxidized endosulfan was found in the kidney and muscle extracts but neither of the two known metabolites nor endosulfan was found in blood or brain extracts. Endosulfan sulfate was the only identified material stored in tissues of mice fed single doses or continuously for 49 days. A compound believed to be the diol was found in urine of mice fed endosulfan, endosulfan sulfate, endosulfan diol, or endosulfan ether. Large amounts of endosulfan sulfate were detected in the liver and traces in the kidney 24 hours after animals received single doses of endosulfan (360).

Endosulfan-^{14}C was fed to milk sheep. Analysis of milk samples showed that up to 88% of the radioactive materials remained in the cream. Gas and thin-layer chromatography showed that the radioactive material was almost entirely endosulfan sulfate. Analyses of urine samples showed the presence of endosulfan alcohol and α-hydroxyendosulfan ether. Other metabolites present were not identified (1728).

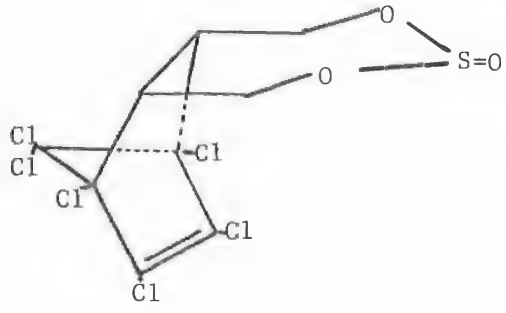
Endosulfan was applied to forage crops on which cattle were allowed to graze beginning seven days after application. Endosulfan sulfate was found in the silage and in the fat of only one animal. Endosulfan I and II were also found in the fat. No detectable residues were found in the milk from dairy cows fed the treated silage (98).

After spraying endosulfan (3:2 mixture of I & II isomers) on foliage, "I" decreased to 30% of initial deposit after one week and to 2% after 3 weeks. Endosulfan II decreased to 47% after one week and to 2% after 7 weeks. All leaf extracts contained a more persistent compound identified as endosulfan sulfate. Seventy-five percent of the residue was in the form of the sulfate after 3 weeks and 90% after 7-11 weeks (618).

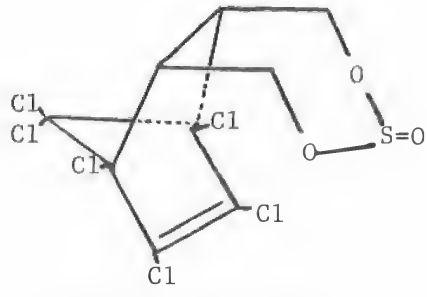
After topical application of C^{14}-endosulfan to Musca domestica (85, 86) or to leafy plants (248), endosulfan sulfate was isolated. Several additional metabolites were also found but not identified. Characteristics of the unidentified compounds indicated that they were probably conjugates of endosulfan as either the glucoside, the glucuronide, or the ethereal sulfate.

After peroral, cutaneous, and subcutaneous application of endosulfan to male imagos of the locust (Pachytilus migratorius migratorioides), four metabolites were observed and identified as: endosulfan sulfate; endosulfan ether; endosulfan hydroxyether; and endosulfan lactone (77).

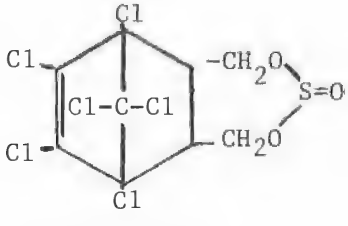
On the basis of NMR and infra-red spectra, the configurations of the two isomers of endothal were assigned (1786).



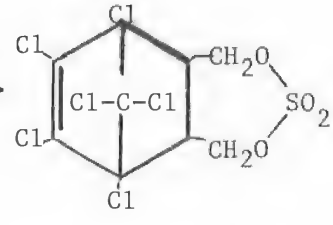
α -Endosulfan (I)



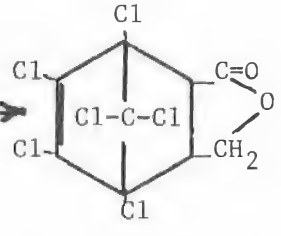
β-Endosulfan (II)



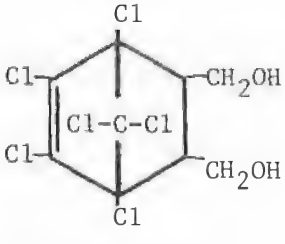
Endosulfan



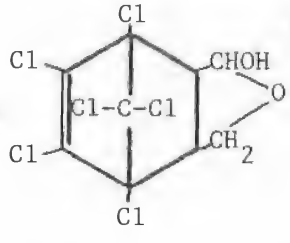
Endosulfan sulfate



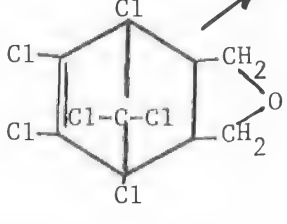
Endosulfan lactone



Endosulfan diol



Endosulfan hydroxyether



Endosulfan ether

Endothal [Disodium 3,6-endoxohexahydrophthalate]

When labeled endothal was fed to adult rats (Wistar strain), over 90% of the radioactivity was recovered in the feces as unchanged endothal. The remainder of the label was recovered unchanged in the urine and as expired CO_2 (1337).

Radioactive tracer studies show that most of the endothal degradation occurred in the moist soil within 7-10 days and that the $C^{14}O_2$ produced is then taken up by the plant and incorporated into plant metabolites (492,1031). Labeled studies indicated at least two unidentified metabolites which were further metabolized. Indications were that the metabolites were organic acids and probably combined with amino acids (1027).

Endothion [2-(S-Dimethylphosphorodithiomethyl)-5-methoxy-4-pyrone]

Endothion was applied to several species of insects and the degree of penetration and metabolism was studied. Three metabolites were isolated but the quantities were insufficient to permit rigorous identification (1419).

After treatment of plants with S^{35}-endothion, the monomethyl derivative was detected (625).

EPN [O-Ethyl O-p-nitrophenyl phenylphosphonothioic acid]

Liver homogenates from several species of mammals, birds and fish were used to study nitroreductase activity. A comparison of the data showed that enzymatic activity varied between classes and among species within a given class of animals. Although reduction of the nitro group to the amino group probably proceeded via the nitroso and hydroxylamino derivatives, the presence of neither one was detected. Species tested included: rat, mouse, guinea pig, chicken, English sparrows, bullhead, sucker, flounder, sculpin, large mouth bass, sunfish, bluegill, alewife. Distribution of nitroreductase in rats varied among tissues: liver > kidney > spleen > heart > lung > erythrocytes > plasma =0. In English sparrows and chickens, activity in kidney exceeded that in liver (669). Rabbit sera brought about removal of sulfur and hydrolysis to p-nitrophenol (1078).

Under culture conditions, B. subtilis reduced EPN to amino EPN (1018).

Ultraviolet irradiation of various formulations of EPN showed decreasing stability in the order: Dust > emulsifiable concentrate > wettable power > pure EPN. Stability also decreased as the concentrations of EPN decreased. The half-lives for 10^{-3}, 5×10^{-4}, and 3×10^{-4} dilutions of 50% emulsifiable concentrate were 3.7, 2.1, and 1.3 hours, respectively (1104).

When EPN was irradiated for 10 hours by ultraviolet, in addition to a resin and unchanged EPN, p-nitrophenol and thiophosphonate were identified by color reactions and infrared spectra. Another compound, which did not possess a P → S linkage, was presumed to be O-ethyl O-(p-nitrophenyl) benzenephosphonate from infrared spectra (1106).

Rabbit sera converted EPN, to p-nitrophenol (1078). After incubation of EPN with $NADPH_2$, oxygen and microsomes of rat, housefly, or rabbit liver, the corresponding phenol was found in each case. The oxon analog was also produced. Some conversion of the oxon to the corresponding phenol was also observed (1700).

EPTC (Ethyl-N,N-di-n-propylthiolcarbamate)

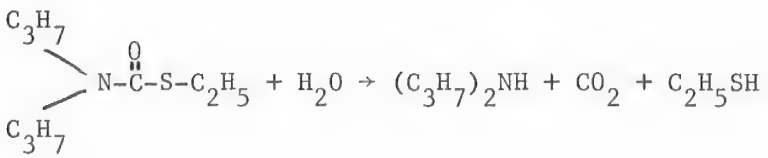
PEBC (Propyl-N,N-ethyl-n-butylthiolcarbamate)

Studies showing a direct relation between the effectiveness of EPTC and the clay and organic matter of the soil indicated that the material was strongly absorbed. The data also suggested that soil microorganisms contributed significantly to the loss of EPTC toxicity when incorporated into soil (435, 495, 917, 1284).

EPTC was hydrolyzed on standing in water. The reaction is first order and has an energy of activation of 7500 calories/mole (1070).

$$k = 2.8 \times 10^{-4} \quad (20°C)$$

$$= 4.25 \times 10^{-4} \quad (30°C)$$



Studies with S^{35}-labeled EPTC showed that this material is readily absorbed from the soil, translocated throughout the entire plant, and rapidly metabolized (433, 434, 1524). No metabolites were identified.

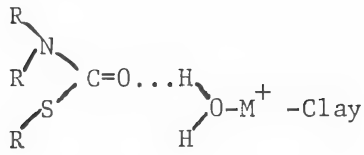
Labeled studies with PEBC showed that the metabolic breakdown in tomato foliage and fruits was a first-order process. The rate constant depended upon the tissue and the growth stage (425).

The mechanism of adsorption of EPTC by montmorillonite was studied with infrared absorption. The results suggested that at least three mechanisms were involved (1042):

(1) Coordination of EPTC to exchangeable metal cations through the carbonyl.

(2) Coordination of EPTC to metal cations through the nitrogen if the molecule is sterically hindered.

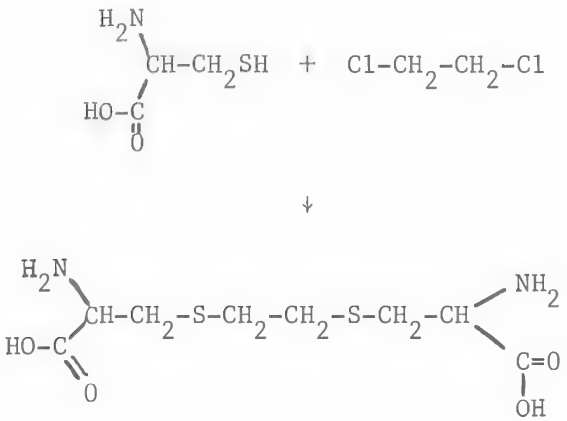
(3) Hydrogen bonding between methylene groups and surface oxygen atoms on the clay mineral surface.



In the presence of an organic cation such as pyridinium, the montmorillonite-EPTC complex exhibited greater stability (1631).

Ethylene bromohydrin [1-Bromo-2-hydroxyethane]
Ethylene dibromide (EDB) [1,2-Dibromoethane]
Ethylene dichloride [1,2-Dichloroethane]

1,2-Dichloroethane reacted with the free sulfhydryl group of cysteine to form the thioether S,S'-ethylenebiscysteine. No chlorine was found in the reaction product. From fish solids treated with 1,2-dichloroethane, a sulfur-containing compound was obtained that exhibited the same R_f as the synthetic thioether S,S'-ethylenebiscysteine; however, isolation and characterization was not carried out (1619).



The metabolism of ethylene dibromide was studied after administration to rats by stomach tube. Three metabolites were isolated from urine and identified: (1) N-acetyl-S-(β-hydroxyethyl) cysteine; (2) S-(β-hydroxyethyl) cysteine; and (3) S-(β-hydroxyethyl) mercapturic acid. The same metabolites were detected in urine after administration of ethylene dichloride or ethylene bromohydrin to rats (1065).

A thorough discussion of the sorption of ethylene dibromide on various soils and the thermodynamics of adsorption by Ca-montmorillonite, Ca-kaolinite, and other soils has been presented (221, 222, 223, 750, 751, 1619, 1620). Sorption coefficients have been tabulated for soils of different organic, moisture and clay content. Temperature coefficients of sorption could be calculated from

$$k_t = k_{15}[1-0.0485(t-15].$$

Temperature coefficients of sorption and the heat of sorption were tabulated (221) and the mechanism of sorption on moist soils investigated (223).

The steady-state diffusion coefficient of ethylene dibromide in soils was related to the free diffusion coefficient.

$$D = 0.066 D_0 (S - 0.1)$$

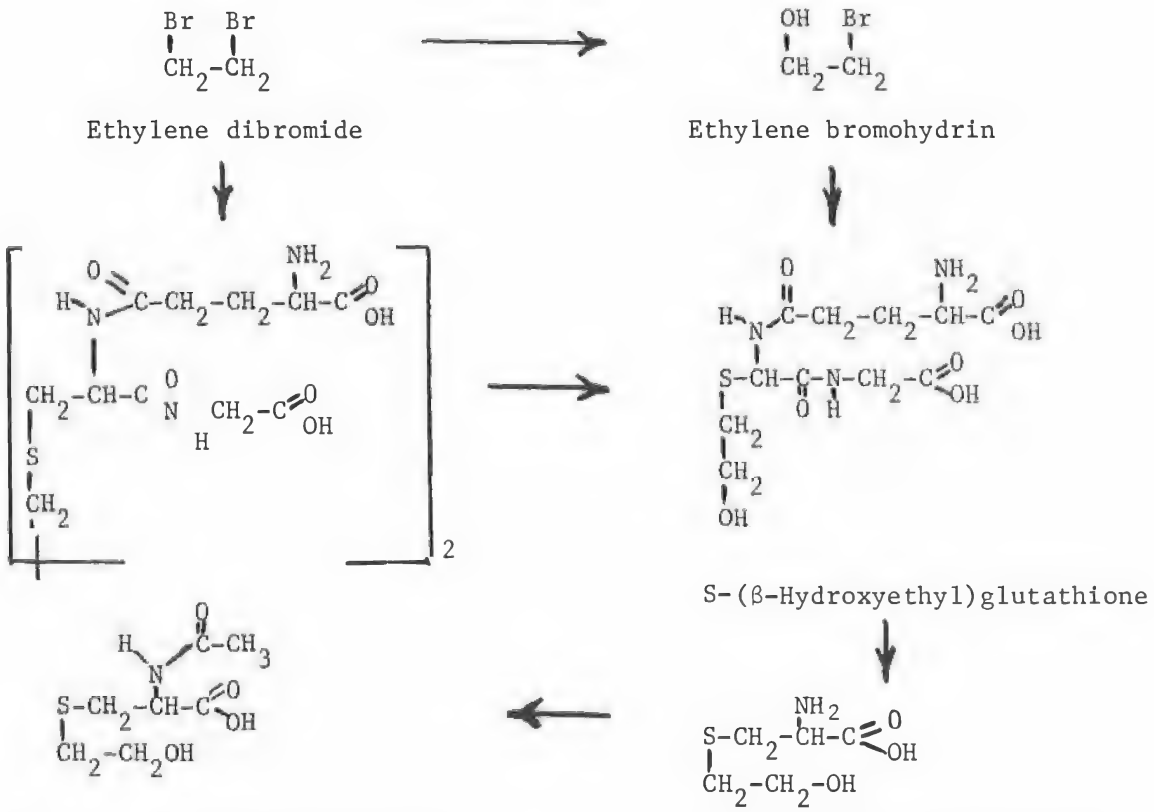
D_0 = diffusion coefficient in free air

S = porosity

Unsteady-state coefficients were calculated from

$$D'' = \frac{S\, D_0}{K(HB+S)}$$

and tabulated (222).



Part of the ethylene dibromide adsorbed on wheat underwent decomposition to ethylene glycol and inorganic bromide on heating. The glycol reacted to a small degree with the protein, probably the $-SCH_3$ of methionine (176).

Ethylene Oxide

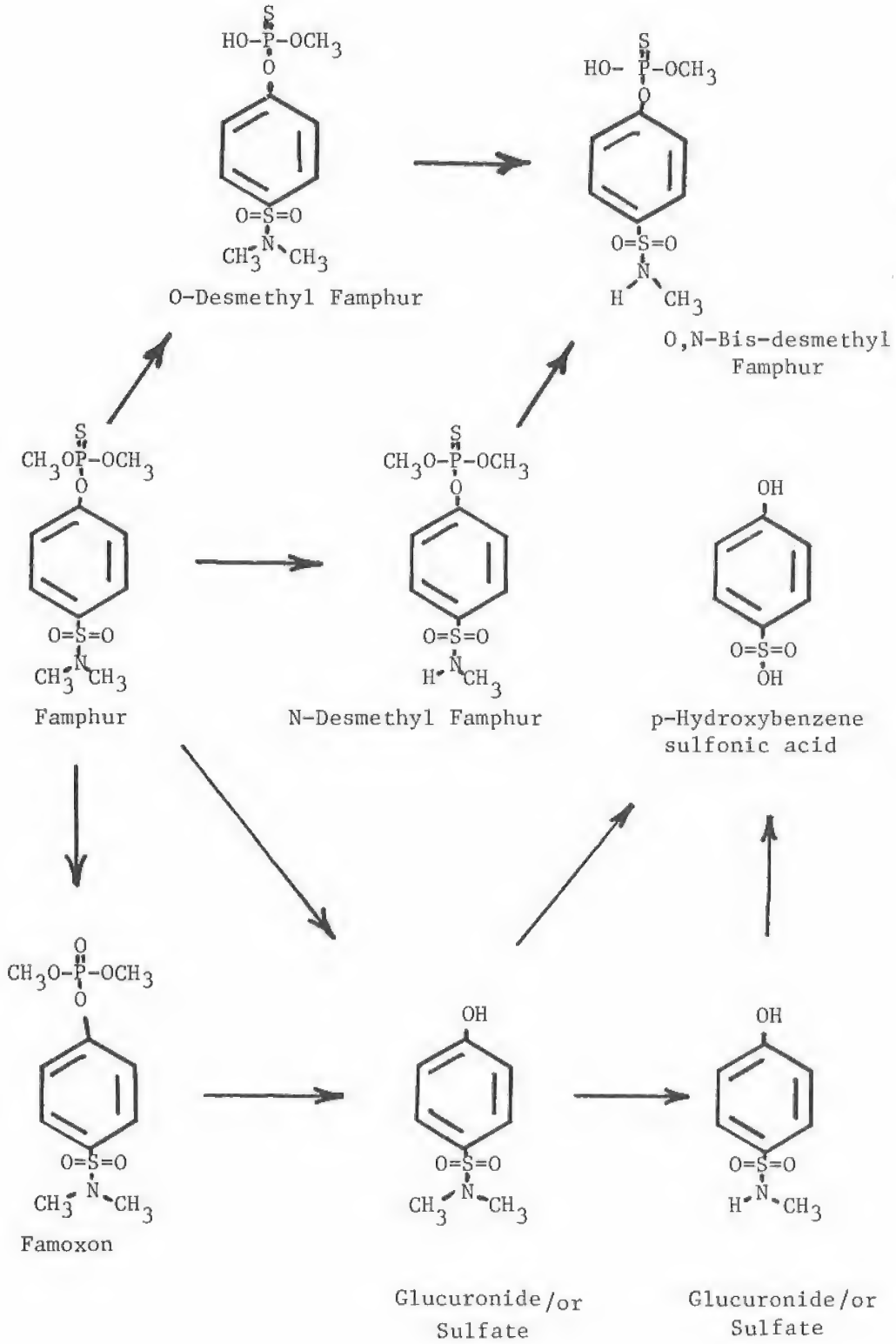
Propylene Oxide

Under conditions used for fumigation of foodstuffs, ethylene oxide and propylene oxide can combine with moisture to form the glycol. In the presence of inorganic chloride of foodstuffs, the corresponding chlorohydrins were formed (1685, 1686).

Famphur [O-(p-N,N-dimethylsulfamoylphenyl) O,O-dimethyl phosphorothioate]

Famphur was degraded at varying rates by mice, American cockroach (Periplaneta americana L.), and milkweed bugs [Oncopeltus fasciatus (Dall.)]. H^3-labeled famphur was injected and analyses were made by thin-layer chromatography. In addition to famphur, both famoxon and N-desmethyl famphur were found (1095).

Two white-faced western ewes and one heifer calf were treated with tritiated famphur. In addition to the oxygen analog, O-desmethyl, O,N-bisdesmethyl, two glucuronides, p-hydroxybenzenesulfonic acid, and several unknown metabolites were observed. The latter may be the mono- or di-dimethylsulfamoylphenyl sulfates. In rats, the major metabolites was dimethylsulfamoylphenyl glucuronide. O,N-bisdesmethyl famphur, methylsulfamoylphenyl glucuronide, and p-hydroxybenzene sulfonic acid were also observed (528).


O-Desmethyl Famphur
O,N-Bis-desmethyl Famphur
Famphur
N-Desmethyl Famphur
p-Hydroxybenzene sulfonic acid
Famoxon
Glucuronide/or Sulfate
Glucuronide/or Sulfate

Fenthion (Bayer 29493, Mercaptophos, Baytex, Lebaycid) [0,0-Dimethyl 0-(4-(methylthio)-m-tolyl) phosphorothioate]

P^{32}-labeled fenthion was applied dermally and intramuscularly to dairy cows. Residues of the fenthion sulfoxide and/or sulfone and/or sulfone of the oxygen analog, as well as the oxygen analog, were present in urine. Dimethyl phosphate and dimethyl phosphorothioate, and an unknown thought to be desmethyl fenthion, were also found in the urine. Similar findings were observed in feces. In edible tissues, results were similar except that phosphorothioic acid was the predominant hydrolysis product (812).

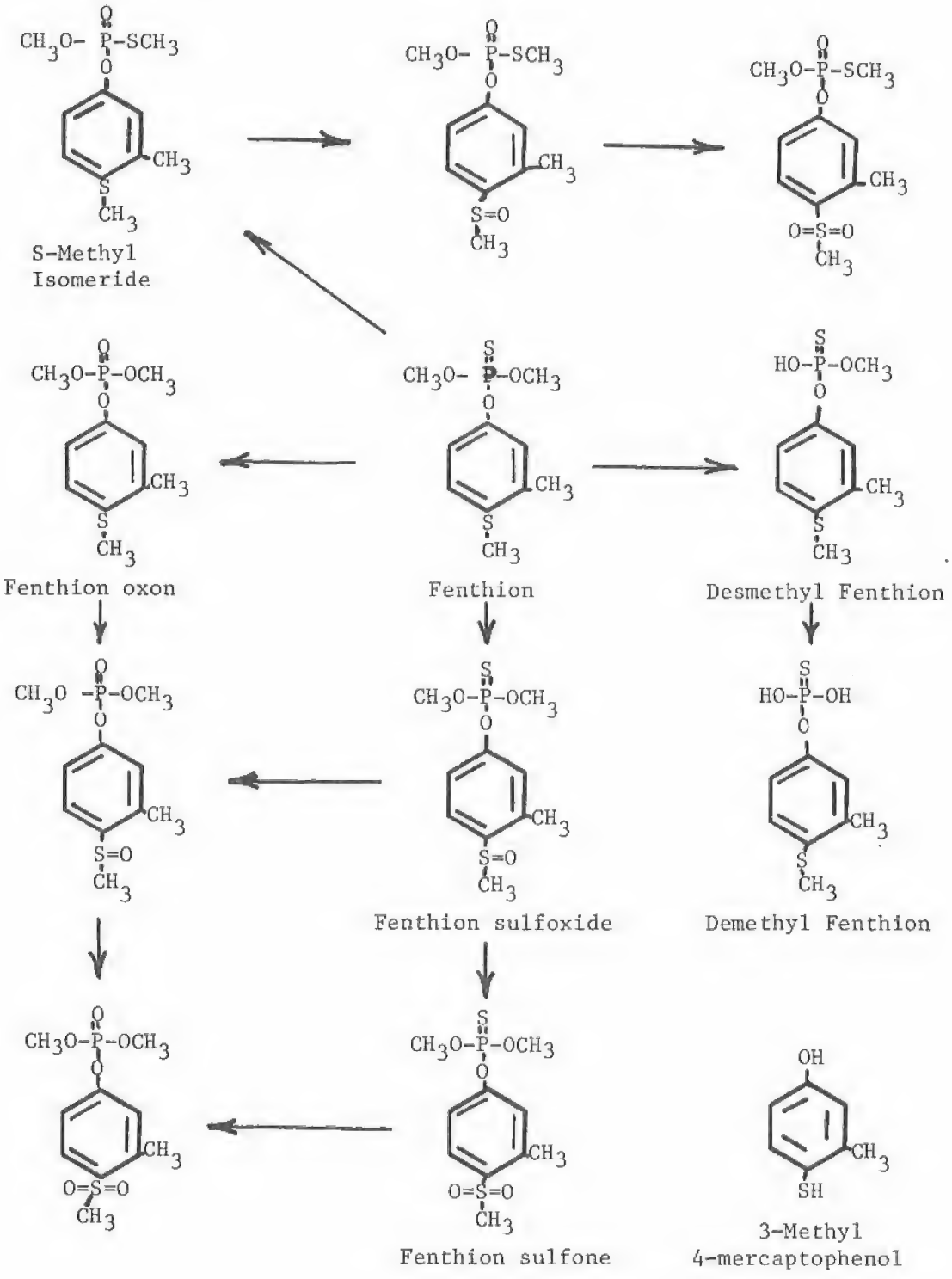
In suicides with fenthion, 3-methyl-4-mercaptophenol has been identified in stomach contents and in urine by means of TLC, paper chromatography, and ultraviolet spectra. No dimethyl phosphorothioate was observed (282).

When rats, houseflies, the German cockroach, boll weevil, and cotton plant were exposed to P^{32}-labeled fenthion, five compounds were found in different proportions (165).

Sulfoxide appeared as the chief metabolite of fenthion when the latter was applied to bean plants. In addition to the sulfoxide, the sulfone, and the sulfoxide and sulfone of the phosphate, and the S-methyl isomeride were also found. The latter was also oxidized to the corresponding sulfone and sulfoxide (479, 1084). When applied to cabbage and tea plants, metabolism was rapid. The sulfoxide and sulfone were the main metabolites although some oxidation of thiophosphate to phosphate also occurred (1413). On rice plants, only about 10% of the applied material remained after six hours. The major part of the metabolites in both rice leaf and ear was the sulfoxide and sulfone. The S-methyl isomer was also found in ears. Using labeled material, almost half of the activity was found in bran; 6.5% in the husk, and 14.7% in polished rice. Fourteen days after application, water soluble metabolites were found in the rice grains: phosphate and thiophosphate, dimethyl phosphate and thiophosphate, demethyl fenthion, and an unknown compound (452).

Exposure of fenthion to ultraviolet light gave rise to a compound believed to be the sulfoxide (1608).

After field treatment of coastal bermudagrass and corn with fenthion, both were analyzed for residues. The bermudagrass contained residues of five metabolites in addition to fenthion: fenthion sulfoxide and sulfone, fenthion oxon and its sulfoxide and sulfone. Corn contained residues of fenthion, fenthion sulfoxide and sulfone, and fenthion oxon sulfoxide and sulfone (1744).


S-Methyl Isomeride
Fenthion oxon
Fenthion
Desmethyl Fenthion
Fenthion sulfoxide
Demethyl Fenthion
Fenthion sulfone
3-Methyl 4-mercaptophenol

Fluoro-Compounds

Fluoroacetamide $[F\text{-}CH_2\text{-}\overset{O}{\overset{\|}{C}}\text{-}NH_2]$

Fluoroacetate $[F\text{-}CH_2\text{-}\overset{O}{\overset{\|}{C}}\text{-}ONa]$

Fluoroacetate and fluoroacetamide are convulsants for the cockroach as well as the mouse. At high doses, hyperexcitability was followed by prostration and tremors. In general, fluoroacetate was more toxic to mice than insects. The reverse was found with fluoroacetamide, probably because of the higher rate of hydrolysis in insects. Fluoroacetamide was hydrolyzed to fluoroacetate. This then entered the tricarboxylic acid cycle where fluorocitrate formed and blocked the citric acid cycle by inhibition of aconitase (938, 1666).

Following injection of fluoroacetate-2-C^{14} into intact rats, about 3% of the label appeared in respiratory CO_2 and 32% in the urine within four days. Labeled fluorocitrate was recovered from the urine. Incorporation of label into cholesterol and into both lower and higher fatty acid fractions was demonstrated. Other unidentified labeled materials were also observed (1665).

When fluoroacetate-2-C^{14} was applied to plants (Acacia geoginae, castor bean, peanut, pinto bean), labeled carbon dioxide was observed. Some label was also incorporated into water soluble fractions and lipids (1177). In some plant seeds, fluoroacetate was converted into fluorine-containing long chain fatty acids. The main constituent apparently was fluorooctadecenoic acid (1667, 1668).

Bacteria have been isolated that are capable of metabolizing both fluoroacetate and fluoroacetamide with release of F^- (780).

A pseuodomonad isolated from Potomac River mud had an enzyme capable of catalyzing the reaction

$$F\text{-}CH_2COO^- + OH^- \rightarrow HOCH_2COO^- + F^-$$

The pH optimum was 9.3 and $K_m = 2.4 \times 10^{-3}$ M. If the reaction proceeded

through a thio ether, then the over-all reaction could be considered in two parts:

$$Enz - S^- + F\text{-}CH_2COO^- \rightarrow Enz\text{-}S\text{-}CH_2COO^- + F^-$$

$$Enz\text{-}S\text{-}CH_2COO^- + OH^- \rightarrow HOCH_2COO^- + Enz\text{-}S^-$$

(557, 558).

Fluoroacetanilides

Studies indicated that monofluoroacetanilides were hydrolyzed in insects, as in warm blooded animals, to yield the corresponding anilines and monofluoroacetic acid. As in warm blooded animals, citrate accumulation was observed in treated insects. Although the mechanism of poisoning was the same in both species, their respective hydrolyzing enzymes were different. The insect enzymes were not so readily inhibited by dipterex and triphenylphosphate (known to be specific inhibitors of the enzyme in warm blooded animals (23).

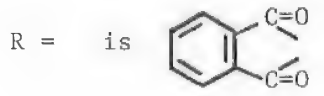
Folpet [N-(Trichloromethylthio)phthalimide]

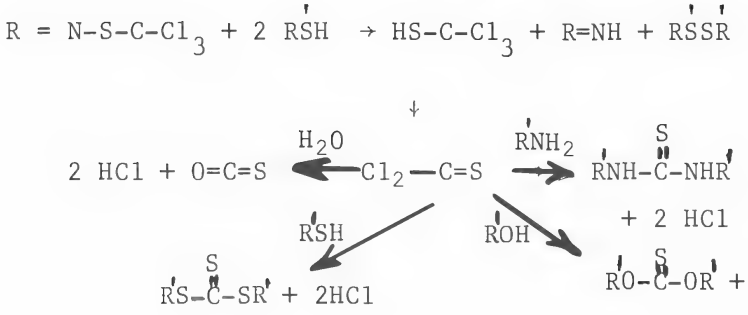
C^{14}- and S^{35}- labeled folpet was used to study the fate of folpet in cultures of Saccharomyces pastorianus. Folpet was rapidly degraded and most of the C^{14}-label could be recovered as phthalimide. S^{35}-label was distributed through the gas phase, culture medium and cells, in both 80% ethanol-soluble and -insoluble fractions (1670).

In studies of the reactions of folpet with proteins, nucleic acids and subscellular components of Saccharomyces pastorianus, sulfur equivalent to 32, 17, 16 and 5 micrograms of folpet was bound per milligram of protein by components of a dialyzed S. pastorianus extract, alcohol dehydrogenase, glyceraldehyde-3-phosphate dehydrogenase and lysozyme, respectively. Very little S^{35} was bound by bovine DNA, yeast RNA or wall fragments of S. pastorianus. Reaction of folpet with cell components could proceed by one of the two paths shown (1671).

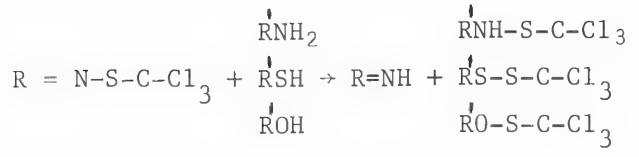
Other studies have shown that folpet reacts with thiols such as cysteine and glutathione. The reaction does not occur below pH 4.0 or 5.0, respectively, and the rate rises rapidly above pH 6.0 (889).

A





B



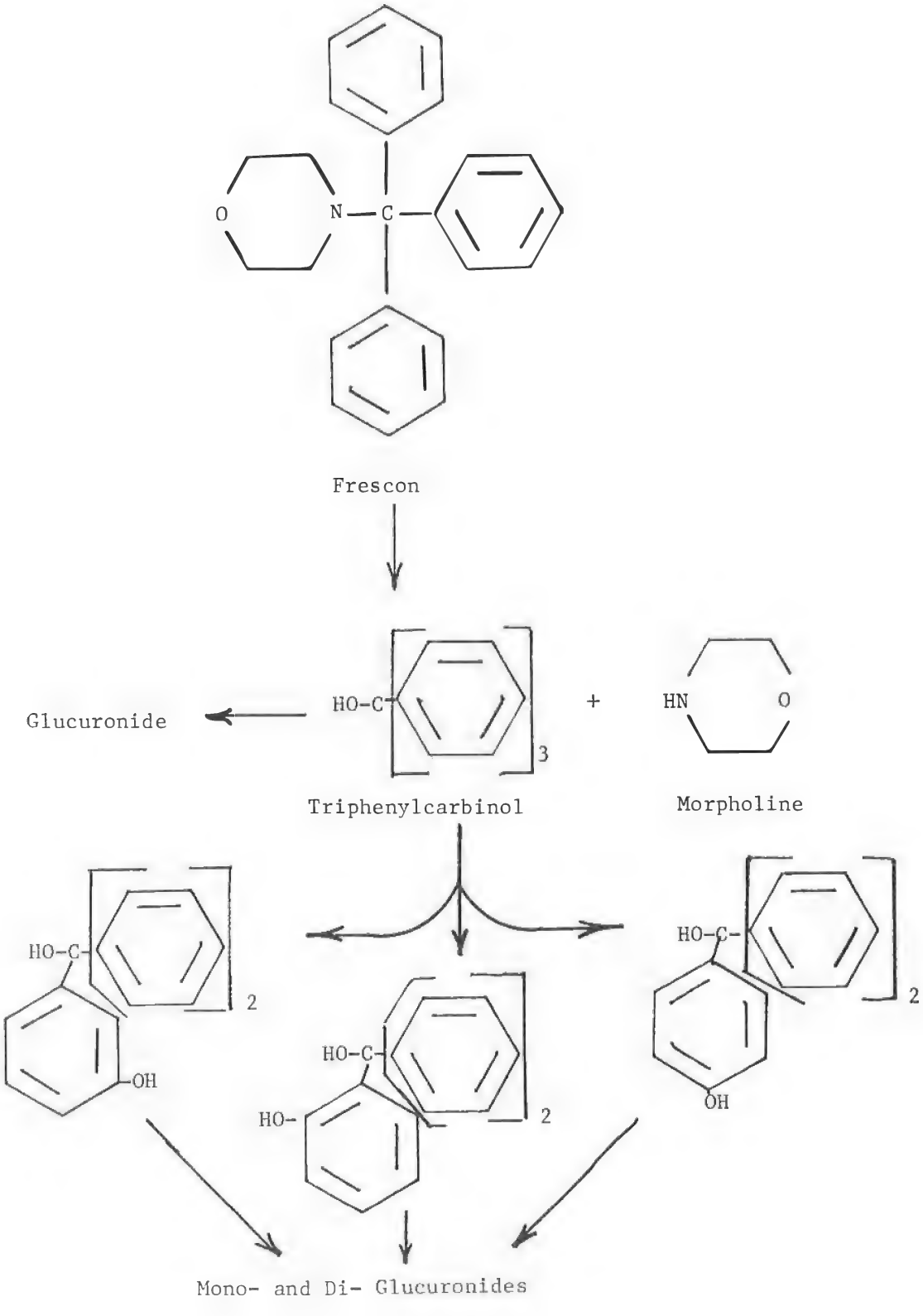
Frescon (W1 8008, Trityl Morpholine) [N-Triphenylmethyl morpholine]

Crops were grown under greenhouse conditions in soil treated with labeled tritylmorpholine. The major metabolite found in rice plants was triphenylmethanol. Some unchanged material and small amounts of the o-, m- and p-hydroxytriphenylcarbinols, free and conjugated, were also observed (113, 114, 115).

In water, N-tritylmorpholine hydrolyzed to triphenylmethanol. The rate was dependent upon pH. At room temperature (22°C) and at initial concentrations of about 0.05 ppm, the pH and half-life are related over the range pH 6-9 (112)

$$\log_{10} (t\ 1/2) = (pH-6)$$

Rats and dogs were given labeled Frescon as single oral doses. No $C^{14}O_2$ was expired and less than 3% of C^{14} remained in carcass and skin after 96 hours. Frescon rapidly hydrolyzed to triphenylcarbinol and morpholine in the stomach. Morpholine was rapidly absorped and excreted largely unchanged though some was degraded. Triphenylcarbinol was absorbed slowly and oxidized to p-hydroxyphenyldiphenylcarbinol; and triphenylcarbinol and its p-hydroxy analog were found in urine, bile and feces, both free and conjugated with glucuronic acid. Traces of ortho and meta hydroxyphenyldiphenylcarbinol were detected both free and conjugated. There appeared to be three glucuronide fractions, possibly diglucuronides of hydroxytriphenylcarbinols, carbinol-group derived glucuronides and phenolic-group derived glucuronides (1734).


O
N
C
Frescon
Glucuronide
HO-C
3
+
HN
O
Triphenylcarbinol
Morpholine
HO-C
2
OH
HO-C
HO-
2
HO-C
2
OH
Mono- and Di- Glucuronides

Furadan (NIA - 10242) [2,2-Dimethyl 2,3-dihydrobenzofuranyl-7-N methylcarbamate]

Male Swiss mice were administered orally a solution of labeled furadan in propylene glycol. Urine was collected and chromatographed. In addition to $C^{14}O_2$, 3-hydroxy furadan (IV) (the major component) and 3-keto furadan (VIII) were identified. After acid hydrolysis of the aqueous portion of ether extracted urine, the 3-keto-furadan phenol (IX), furadan phenol (VI), 3-hydroxy furadan (IV) and two unidentified compounds were found (990).

In other studies with laboratory rats, after oral administration of labeled furadan in Tween 20, six metabolites were identified. Analyses also indicated the presence of conjugates and three unidentified compounds. Found and identified were: 3-hydroxy-N-hydroxymethyl furadan (III) and its conjugate (XI); N-hydroxymethyl furadan (II); 3-hydroxy furadan (IV) and its conjugate (V); 3-keto furadan (VIII); 3-hydroxy furadan phenol as a conjugate (XIII); 3-keto furadan phenol (IX) and its conjugate (XIV); and furadan phenol (VI) and its conjugate (XII) (375).

Incubation of furadan with rat liver homogenates gave rise to compounds II, III, IV, VI, VII, IX, one unidentified compound and some water soluble materials (375).

When exposed to furadan, the dairy cow excreted 12, 1.0 and 2.6% of the carbonyl-C^{14} label in urine, feces, and milk, respectively. Ring-C^{14} was excreted to the extent of 83, 2.9 and 0.5%, respectively. In the urine, seven metabolites were identified: 3-hydroxy furadan (IV) (5%); 2,3-dihydro-2,2-dimethyl-7-benzofuranyl sulfate (XX) (38%) and glucuronide (XII) (7%); 2,3-dihydro-2,2-dimethyl-3-oxo-7-benzofuranyl sulfate (XXII) (9%) and glucuronide (XIV) (1%); 2,3-dihydro-2,2-dimethyl-3-hydroxy-7-benzofuranyl sulfate (XXI) (3%) and glucuronide (XIII) (12.5%).

In other studies, after administration of furadan to a cow, about 0.2% of the dose was eliminated in milk, 0.7% in the feces and 94% in the urine. Analysis of the milk revealed the presence of 3-hydroxyfuradan, 3-hydroxy-N-hydroxymethylfuradan, 3-keto furadan, 3-hydroxyfuradan phenol and an unidentified compound. These were present in both the free and conjugated forms. In the urine, the following metabolites were found: 3-hydroxyfuradan, 3-hydroxy-N-hydroxymethylfuradan, 3-hydroxyfuradan phenol, 3-keto-furadan phenol and furadan phenol. In the feces, the pattern of metabolites was similar to that observed in the urine except that 3-keto furadan was found but 3-keto furadan phenol was not present (1694).

In alfalfa, labeled furadan was metabolized to free and acid labile conjugates of 3-hydroxy furadan (IV) (45%); 2,3-dihydro-7-hydroxy-2,2-dimethylbenzofuran (VI) (5%); 2,3-dihydro-7-hydroxy-2,2-dimethyl-3-oxo

benzofuran (IX) (21%); and 2,3-dihydro-3,7-dihydroxy-2,2-dimethylbenzo furan (VII) (15%). In the cow, 3-hydroxy furadan (IV) was metabolized and excreted as the glucuronide (V) (10%). The carbonyl-C^{14} label from alfalfa conjugates of 3-hydroxy furadan was excreted in urine (38%), feces (18%) and in milk (2.6%) (1641).

Following topical application of furadan to houseflies, there was evidence of formation of only trace amounts of N-CH_2OH derivatives (990). The major metabolite observed was 3-hydroxyfuradan, free and conjugated. 3-keto furadan, 3-hydroxy-N-hydroxymethyl furadan and its conjugate, and N-hydroxy furadan and its conjugate were also identified (375). In other studies with houseflies (Musca domestica), $C^{14}O_2$ was obtained from N-$C^{14}H_3$ as well as from C =O labeled furadan (1001).

In vitro oxidation by mixed function oxidases of housefly microsomes converted furadan into twelve metabolites. Of these, nine were characterized: a 3-keto-dihydroxyfuradan (XVI); 3-keto-6-hydroxyfuradan (X); 3-hydroxy furadan (VI); 3-keto furadan (VIII); 2-hydroxymethyl-3-keto furadan (XVIII); 3-hydroxy-N-hydroxymethyl furadan (III); 3-keto furadan phenol (IX); furadan phenol (VI); and 2-hydroxymethyl-3-keto furadan phenol (XIX) (990).

After ingestion of furadan by the salt marsh caterpillar, Estigmene acrea, compounds IV, V, VI, VII, VIII, IX, XIII and XIV were identified (990).

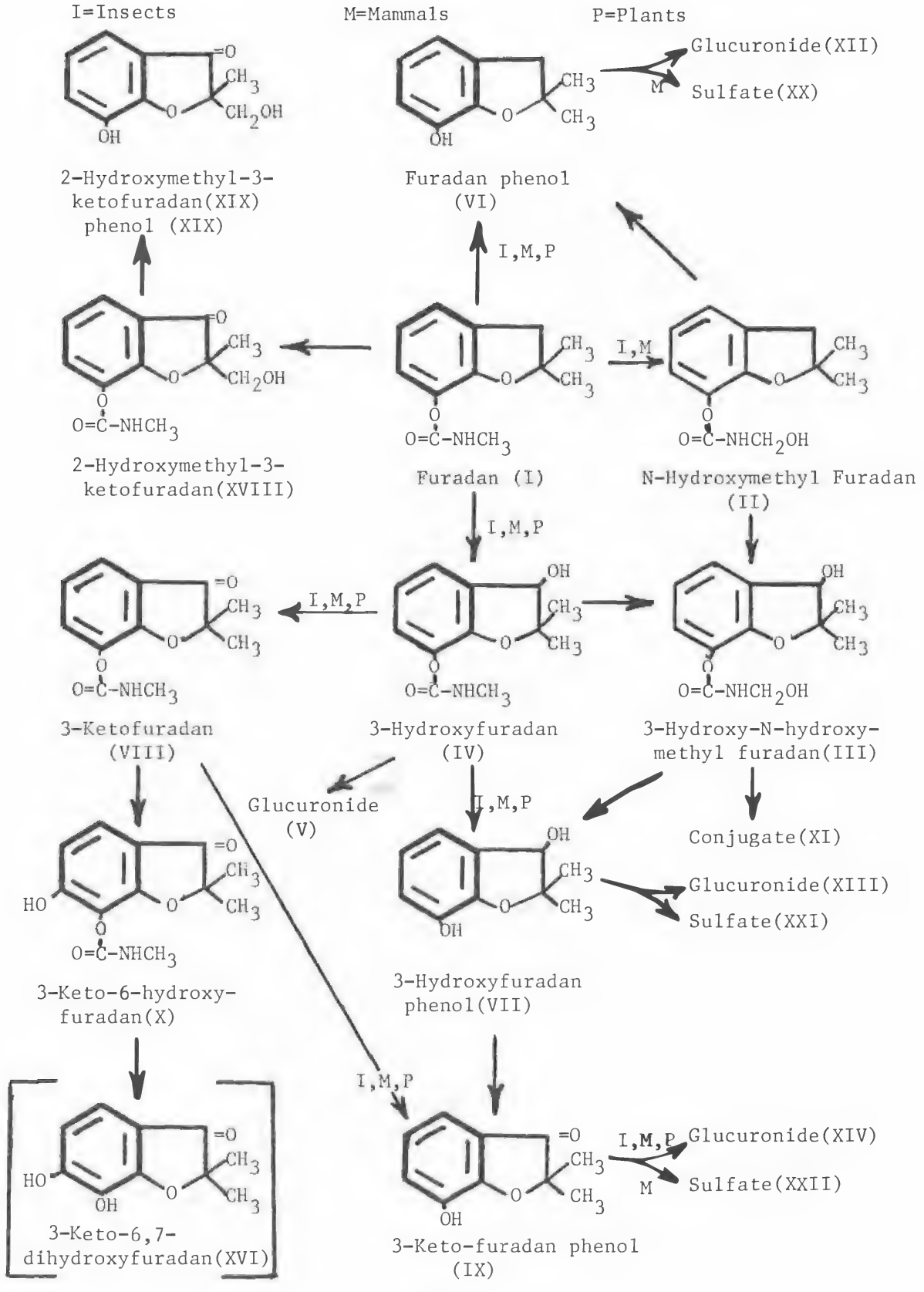
Radiolabeled furadan carbonyl-C^{14} and ring-C^{14}, was injected into the stem of young bean plants (Garden snapbeans, Contender variety). Thin layer chromatography was used to identify the metabolites. Found free and as a water soluble conjugate were: 3-hydroxy-N-hydroxymethyl furadan (III); 3-keto furadan (VIII); 3-hydroxy furadan phenol (VII). Found conjugated only were 3-hydroxyfuradan (IV) and 3-keto furadan phenol (IX). Furadan phenol (VI) was observed only as the free compound. Separation of the conjugates into two bands by TLC indicated that two naturally occurring compounds were conjugating the metabolites (1563).

Exposure of intact cotton plants to furadan gave rise to 3-hydroxy-furadan which was oxidized readily to the keto analog. This was followed by hydrolysis and conjugation of the resulting phenols. A compound believed to be the conjugate of 3-keto-6-hydroxy-furadan was also observed (990).

In isolated cotton leaves treated with labeled furadan, furadan phenol (VI) and its glucoside (XII), 3-hydroxyfuradan (IV) and its glucoside (V), 3-keto furadan (VIII), 3-hydroxy furadan phenol (VII) and its glucoside (XIII), 3-ketofuradan phenol (IX) and its glucoside (XIV) and 6-hydroxy-3-keto furadan (X) were identified (990).

Studies were also conducted with corn. In 10-day old corn seedlings cut off above the roots, metabolites IV, V, VI, VIII, IX, XII, XIII and XIV were found; in corn leaves, metabolites IV, V, IX and XIV; and in roots, metabolites, IV, VI and VIII (990).

When furadan was exposed to sunlight, decomposition gave rise to 3-hydroxy furadan (IV), furadan phenol (VI) and two unidentified compounds (990).


I=Insects
M=Mammals
P=Plants
Glucuronide(XII)
Sulfate(XX)
2-Hydroxymethyl-3-ketofuradan(XIX) phenol (XIX)
Furadan phenol (VI)
I,M,P
I,M
2-Hydroxymethyl-3-ketofuradan(XVIII)
Furadan (I)
N-Hydroxymethyl Furadan (II)
I,M,P
I,M,P
3-Ketofuradan (VIII)
3-Hydroxyfuradan (IV)
3-Hydroxy-N-hydroxy-methyl furadan(III)
Glucuronide (V)
I,M,P
Conjugate(XI)
Glucuronide(XIII)
Sulfate(XXI)
3-Keto-6-hydroxy-furadan(X)
3-Hydroxyfuradan phenol(VII)
I,M,P
I,M,P
Glucuronide(XIV)
Sulfate(XXII)
3-Keto-6,7-dihydroxyfuradan(XVI)
3-Keto-furadan phenol (IX)

Griseofulvin (7-chloro-4:6:2'-trimethoxy-6'-methylgris-2'-en-3:4'-dione) [7-chloro-4,6-dimethyoxycoumaran-3-one-2-spiro-1'-(2'-methoxy-6'-methylcyclohex-2'-en-4'-one)]

Cl^{36}-Labeled griseofulvin was administered orally to rabbits, rats and humans. Examination of urine samples indicated that both 4- and 6-demethylgriseofulvin were excreted. These were present as free compounds as well as conjugates. In rats, the relative amounts of these two compounds changed with time, with 4-demethyl analog increasing and the 6-demethyl analog decreasing. The latter was excreted primarily in the free form while 4-demethylgriseofulvin appeared largely in the conjugated form. In vitro studies produced the same metabolites (84, 1380, 1381, 1382). In other studies, the metabolite 3-chloro-4,6-dimethoxysalicylic acid was observed in rabbit urine (1417).

During studies on the enzymatic destruction of griseofulvin by fungi, three different monodemethylgriseofulvins were demonstrated. Microsporum canis produced 7-chloro-4-hydroxy-6,2'-dimethoxy-6'-methylgris-2'-en-3,4'-dione (4-demethylgriseofulvin); Botrytis allii produced griseofulvic acid (2'-demethylgriseofulvin); and Cercospora melonis gave rise to 7-chloro-6-hydroxy-4,2'-dimethoxy-6'-methylgris-2'-en-3,4-dione (6-demethylgriseofulvin) (141).

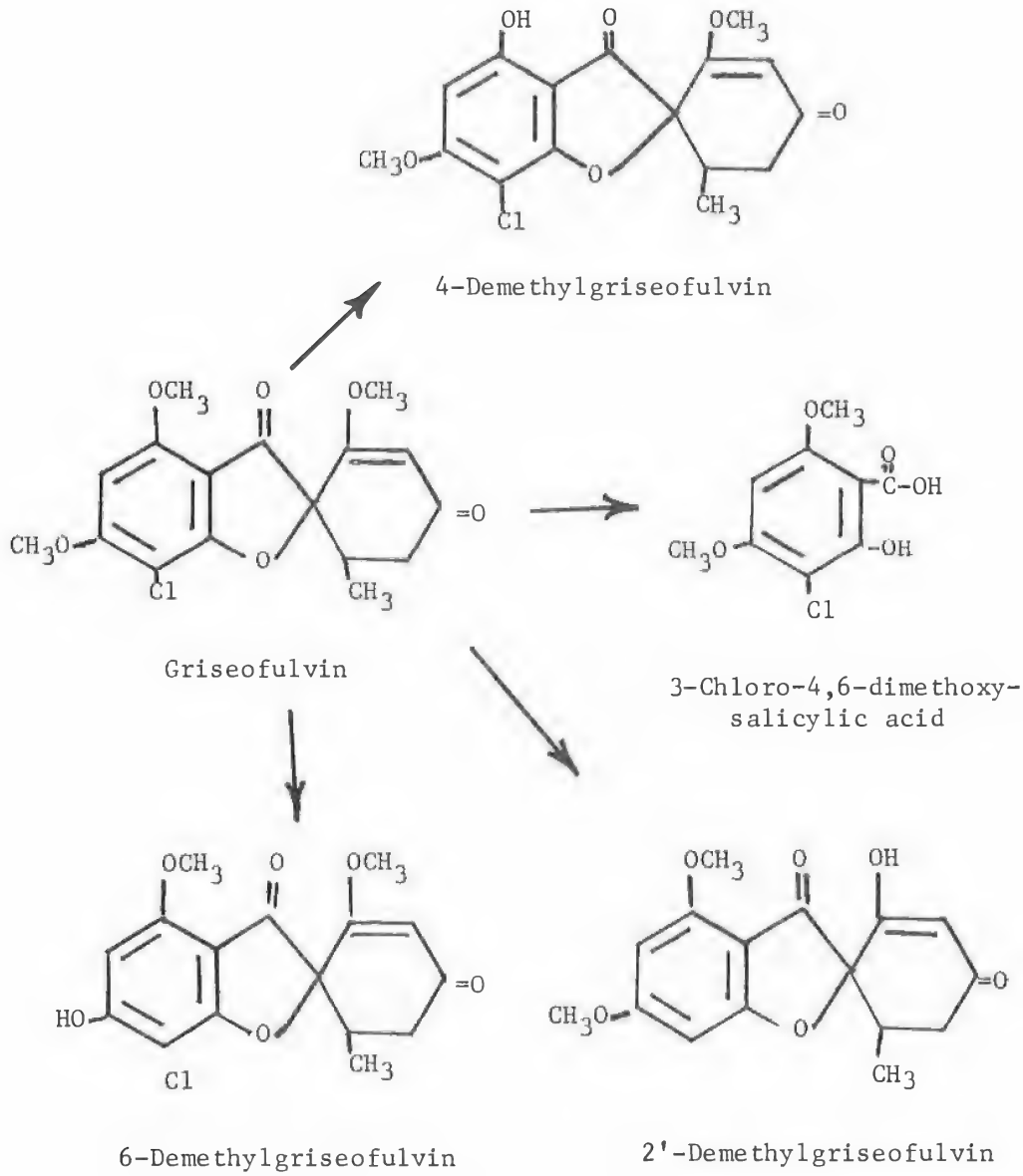
Studies with a pseudomonas gave evidence that griseofulvin was dechlorinated and that ring rupture occurred. No metabolites were identified (1523).

The elucidation of the complex structure of griseofulvin was covered in a series of four papers (565, 566, 567, 568).

Absorption kinetics of griseofulvin in man were studied. The plasma-concentration curve was described by a bi-exponential equation; the half-life for the first exponent was 0.70 - 1.7 hr. and the second 9.5 - 2.1 hr. Absorption occurred up to 30 hours after administration and 27 - 72.5% of the dose was absorbed (1635).

In studies with dogs, 6-demethylgriseofulvin was the primary metabolite. This was excreted in the urine in the free form (40-57%) and as the glucuronic acid conjugate (25%). Only a small amount of the 6-demethyl analog was found in the bile (6%). The plasma disappearance curves of griseofulvin in dogs after intravenous injection were

biexponential. In two dogs, the average half-lives for the fast and slow components were 4 and 47 minutes, respectively (1777).



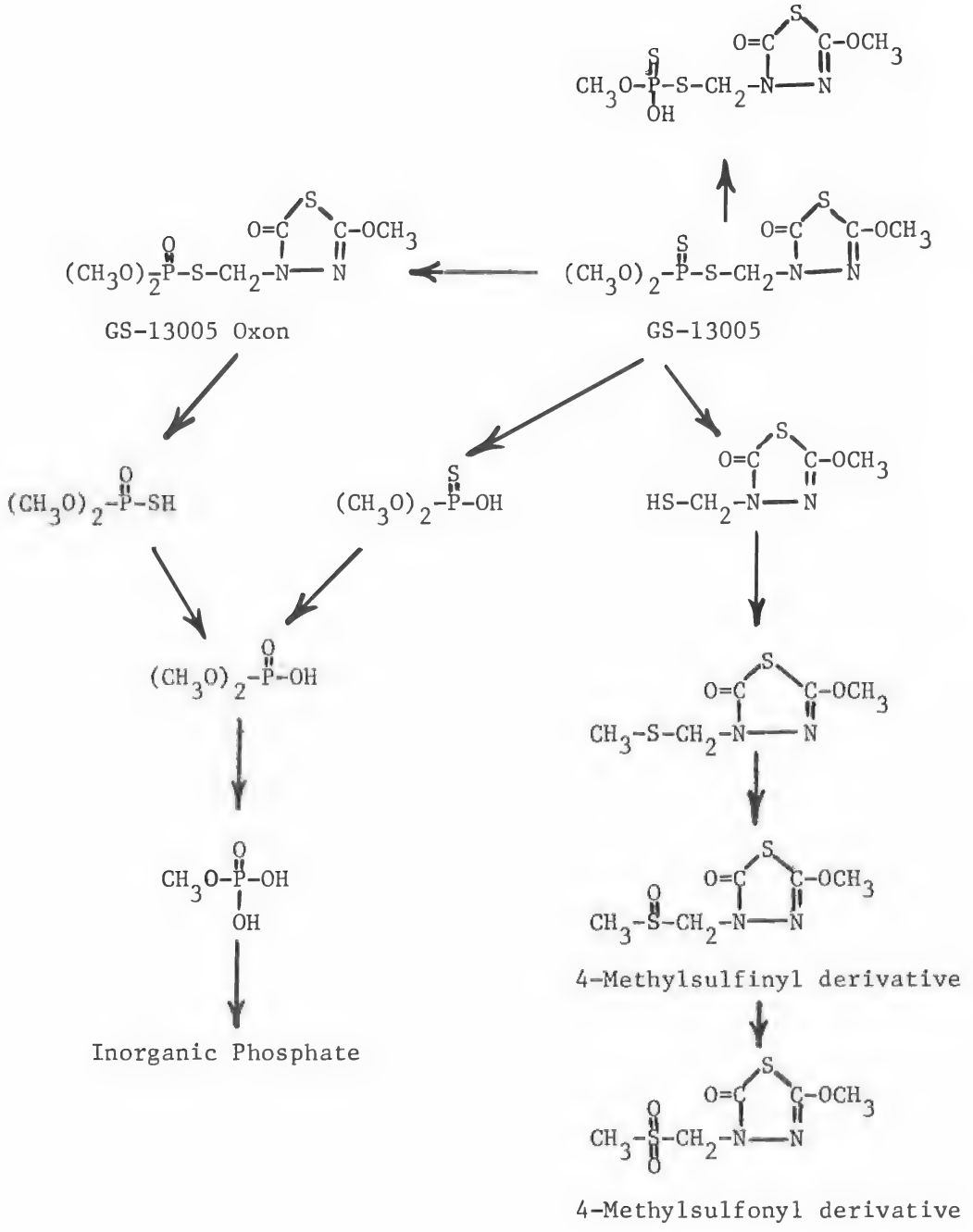
GS-13005 [0,0-dimethyl S-[2-methoxy-1,3,4-thiadiazol-5-(4H)onyl-(4)methyl]-dithiophosphate]

Rats were dosed orally with labeled GS-13005. Distribution patterns indicated complete absorption and intense metabolism of the compound with cleavage of the heterocyclic to CO_2 as the main result. GS-13005 was distributed rapidly but the radioactive content of all organs was below the limit of detection 48 hours after dosage except for traces in the muscles. Most of the dose was excreted in the urine as polar metabolites or expired. The most important metabolites observed were 4-methylsulfinylmethyl- and 4-methylsulfonyl-2-methoxy-1,3,4-thiadiazole-5-one. These metabolites originate by methylation and oxidation of the mercaptomethyl derivative liberated after hydrolysis of the P-S bond (418, 1566). Dimethyl phosphate, dimethyl phosphorothioate, methyl phosphate, inorganic phosphate and desmethyl GS-13005 were also found in urine and/or feces (1629).

Young bean plants (Phaseolus vulgaris L.), to which GS-13005 had been applied, also split the heterocyclic moiety of the insecticide to CO_2. Small amounts of unidentified metabolites were also detected (418).

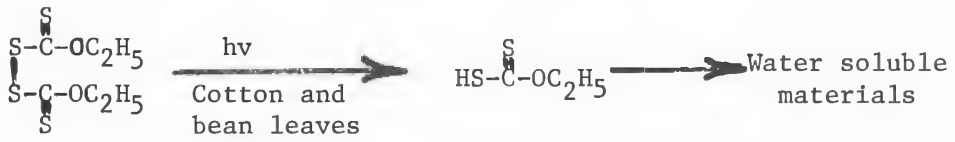
Cotton plants of the Delta-pine Smoothleaf variety were grown and treated with P^{32}-labeled GS-13005. Examination of various parts of the plants revealed the presence, in varying amounts, of desmethyl GS-13005, the oxon (or thiolate) analog of GS-13005, dimethyl phosphorothiolate, mon- and di-methyl phosphate, inorganic phosphate, an unidentified metabolite, and some unextracted radioactivity. The latter probably represented incorporation of liberated inorganic P^{32} into natural plant constituents (1629).

In 5th instar tobacco budworms, GS-13005 was completely metabolized during the first 4 hours. Small amounts of the thiolate analog and the desmethyl analog formed during this time but was depleted after 4 hours. Dimethyl phosphorothioate and dimethyl phosphate were the major hydrolytic products detected in internal extracts and excreta. Other unidentified polar metabolites were also observed (1629).


GS-13005 Oxon
GS-13005
Inorganic Phosphate
4-Methylsulfinyl derivative
4-Methylsulfonyl derivative

Herbisan (Bext-Defoliant, bis(ethylxanthic)disulfide) [Ethyl xanthogen]

In leaves of cotton and bean plants, Herbisan decomposed to xanthic acid and unidentified water soluble materials. This was accelerated by light (834).



2-Heptadecyl-2-imidazoline

Studies aimed at elucidating the mechanism by which this material exerts its fungistatic activity indicated that 2-heptadecyl-2-imidazoline was a competitive inhibitor in the synthesis of the purines guanine and xanthine in Sclerotinia fructicola (1491).

HRS-1422 [3,5-Diisopropylphenyl methylcarbamate]

After exposure of rats to labeled HRS-1422, $^{14}CO_2$ was observed (836). When incubated with rat liver microsomes + $NADPH_2$, HRS-1422 gave rise to the N-hydroxy analog and a number of unidentified metabolites (1110, 1111, 1112).

After exposure to UV (2537 A) , five compounds were observed. Treatment of bean plants also gave rise to several unidentified compounds (3).

See also UC-10854.

Imidan [0,0-Dimethyl S-phthalimidomethyl phosphorodithioate]

After dermal application, imidan was moderately absorbed by the skin of a steer. Approximately 9.6% of the applied dose was recovered in excreta within 7 days and less than 2% from blood. The principal route of elimination was via the urine in which almost 8% of the applied dose was recovered within 7 days. Paper chromatography and electrophoresis indicated that the primary metabolic products were phthalamic and phthalic acids. The presence of benzoic acid, a decarboxylation product of phthalic acid, was indicated also but not conclusively proven (263).

In rats, 79% of the administered dose was eliminated via the urine and 19% via the feces. About 2.6% remained in tissues. Hydrolysis predominated as in the steer and cotton plant (465). After oral administration of labeled imidan to rats, the aqueous fraction remaining after extraction of urine with benzene contained 98% of the labeled imidan metabolites excreted in urine. Using paper chromatography, the following metabolites were found: phthalic acid and a derivative, phthalamic acid, and about eight other unidentified compounds. In the benzene fraction, imidoxon, N-hydroxymethylphthalimide and phthalimide were observed. After acid hydrolysis of metabolites in the benzene and aqueous fractions of urine, chromatography by paper and TLC showed the presence of phthalic acid and 3- and 4-hydroxyphthalic acid (1690).

In the rat feces, imidoxon and unchanged imidan were found. Indications of phthamic acid was also observed. Hydrolysis of aqueous extracts converted all metabolites to phthalamic acid (1690).

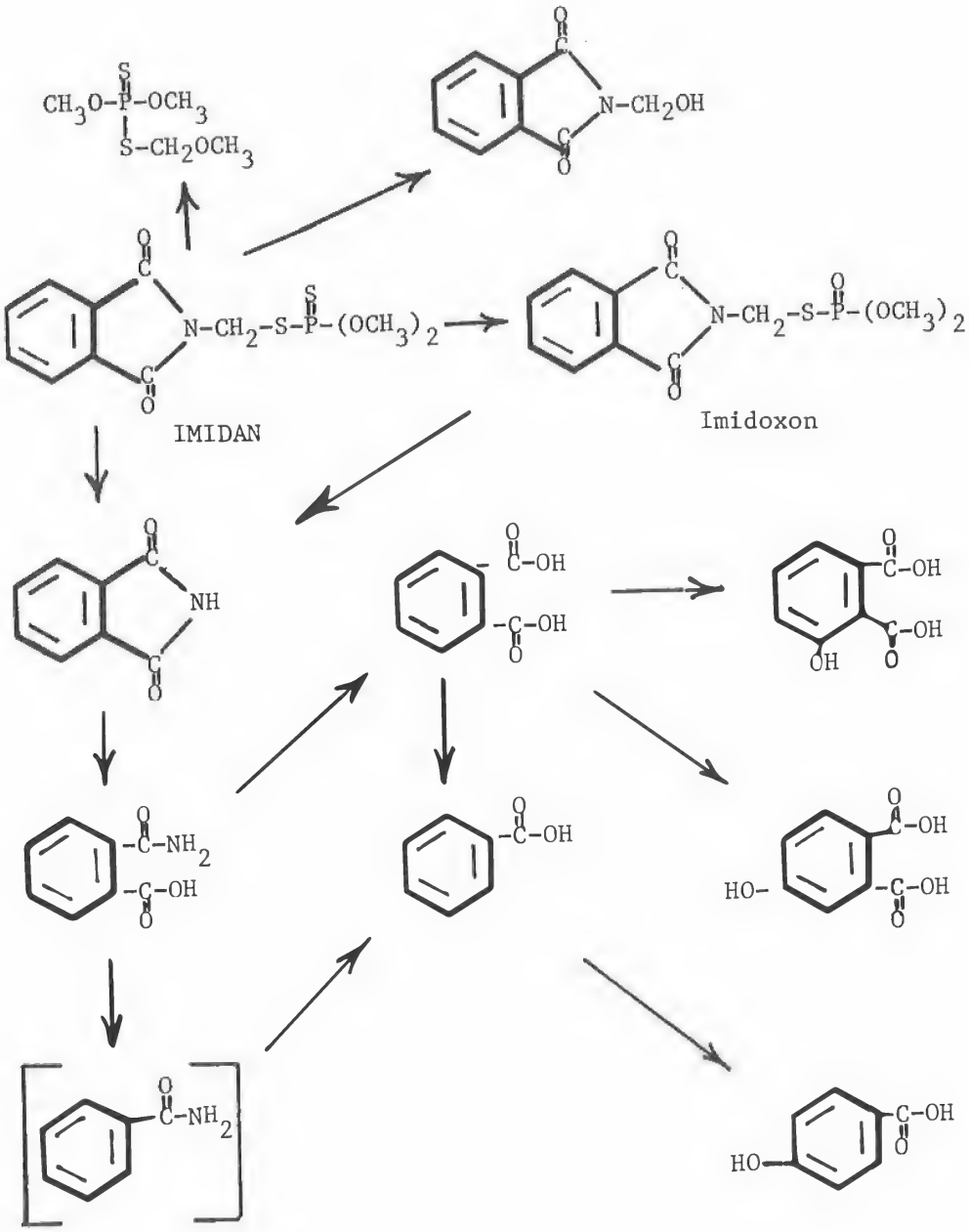
Incubation of imidan with a rat liver microsome system, in the presence of $NADPH_2$ gave rise to four metabolites not identified (1690).

After application of imidan to German cockroaches, imidoxon and phthalamic acid are indicated by paper chromatography. Hydrolysis of material not soluble in acetone converted the labeled material to phthalic acid (1690).

Phthalamic and phthalic acids were found as metabolic or hydrolysis end products also in water, the German cockroach [Blatella germanica (L.)], and the rat (980). When incubated with fly homogenate and $NADPH_2$, imidan gave rise to its oxygen analog and other unidentified compounds (1427).

Hydrolysis of imidan occurred in soils; and tests indicated that this was not dependent on moisture alone but was due in some degree to microbial action. In dry sandy loam soil, the time for 50% degradation was 19 days, compared to 3 days in moist soil (979).

Cotton leaves were treated with C^{14}-imidan. The material was readily absorbed and translocated in the plant. The major non-phosphate metabolites isolated consisted of phthalic and/or phthalamic acid, benzoic acid and p-hydroxybenzoic acid. The thiol analog was not found and the presence of post-hydrolysis O,O-dimethyl methoxymethylphosphorodithioate indicated hydrolysis predominated over oxidation (978).



Ioxynil [3,5-Diiodo-4-hydroxybenzonitrile]

Bromoxynil [3,5-Dibromo-4-hydroxybenzonitrile]

Photolysis of 3,5-diiodo-4-hydroxybenzonitrile (ioxynil) was a factor in its herbicidal action. The herbicidal and molluscicidal actions of ioxynil and bromoxynil, the bromine analog, are more toxic in light than in dark. When irradiated with ultraviolet light for 20 hours, in benzene, ioxynil was converted into 3,5-diphenyl-4-hydroxy-benzonitrile. The corresponding monoiodo derivative gave 3-phenyl-5-hydroxybenzonitrile. Within tissues of plants, ioxynil probably liberates free radicals which interfere with plant functions (1440).

After ioxynil was fed to a cow, a compound was isolated from the urine that appeared to be a conjugate of 3-iodo-4-hydroxybenzonitrile (459).

In acid soils, ioxynil was precipitated. 3,5-diiodo-4-hydroxybenzamide, the benzoic acid analog and traces of iodide were found. In sterile soils this does not occur (1535).

Bromoxynil, fed to Holstein cows, was excreted intact and as an unidentified conjugate in urine (1258).

Isolan [1-Isopropyl-3-methyl-5-pyrazolyl dimethylcarbamate]

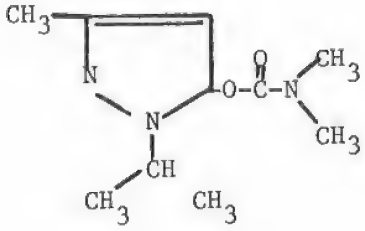
Pyrolan [1-Phenyl-3-methyl-5-pyrazolyl dimethylcarbamate]

After exposure of rats to labeled isolan, $C^{14}O_2$ was found (836). When incubated with a rat liver microsome-$NADPH_2$ system, isolan was degraded to 1-isopropyl-3-methylpyrazolone and a number of unidentified compounds (1110, 1111, 1112).

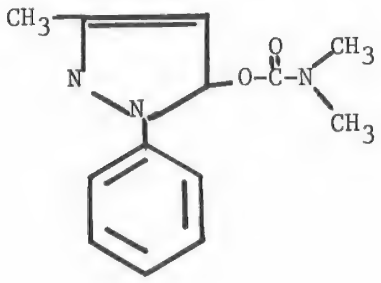
It has been shown that microsomal enzymes, requiring TPNH and oxygen, act on N,N-dialkylcarbamates. Dealkylation occurs with formation of formaldehyde from N,N-dimethylcarbamates. In such a system isolan was degraded and formaldehyde was released at acid, but not neutral, pH. Pyrolan reacted similarly (677).

Exposure of isolan to ultraviolet radiation showed formation of 1-isopropyl-3-methyl-pyrazolone and an unidentified compound. Similar treatment of pyrolan gave rise to 1-phenyl-3-methylpyrazolone. Three unidentified compounds were also observed after prolonged irradiation (391).

After about 75 - 100 days, isolan was completely lost from soils (579).



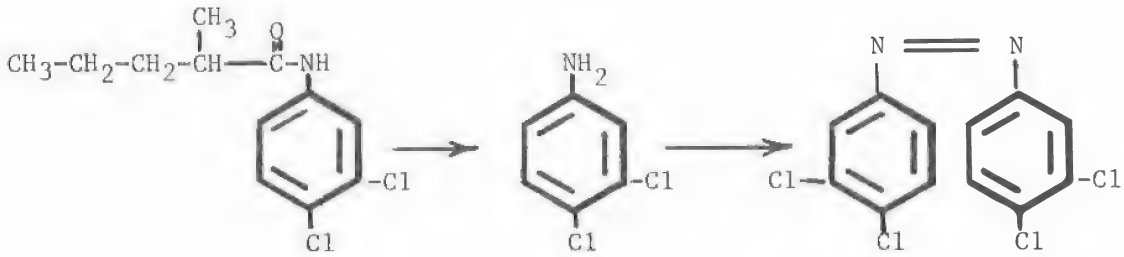
Isolan



Pyrolan

Karsil [N-(3,4-Dichlorophenyl)-2-methylpentamide]

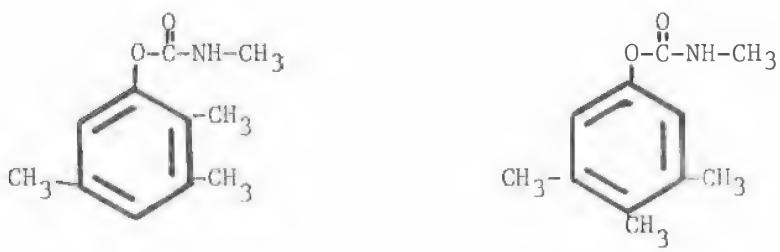
Residues isolated from soil samples that were treated with Karsil and incubated for 2 weeks were identified as dichloroaniline and tetrachloroazobenzene by gas and thin-layer chromatography, melting points, and infrared spectra. Red-brown residues present in extracts of treated soils probably represented complex polyaromatic condensation products. In soil that was sterilized or treated with the metabolic inhibitors NaN_3 and $HgCl_2$, gas chromatographic analyses did not detect aniline or azo compounds. It was concluded that degradation of Karsil was the result of a series of biochemical transformations mediated by soil microorganisms (1627).



Karsil Dichloroaniline Tetrachloroazobenzene

Landrin [2,3,5- and 3,4,5-Trimethyl N-methylphenylcarbamate]

Metabolism of the two isomers of Landrin was similar on bean plants. The major isomer (3,4,5-analog) was degraded to N-hydroxymethyl landrin, the 4-hydroxymethyl and possibly the 3-hydroxymethyl analogs. The 2,3,5-isomer was degraded to the N-hydroxymethyl and the 3-hydroxymethyl derivatives. Glucuronides of the 3- and 4-hydroxymethyl derivatives were also observed (1320).



2,3,5-Landrin 3,4,5-Landrin

Malathion [0,0-dimethyl S-(1,2-bis-carbethoxy) ethyl phosphorodithioate]

Accumulation of malathion or its metabolites in the tissues of hens fed labeled malathion was slow. Two to three per cent was excreted unchanged or as chloroform-soluble metabolites. Ninety-seven to ninety-eight percent is excreted as ionic water-soluble metabolites. Of the tissues examined, liver and kidney contained the greatest amount of radioactive compounds. The level of radioactivity remained constant for a week after withdrawal from the malathion-fortified feed and then slowly decreased. The amount of radioactive compounds decreased more rapidly in the white than in the yolk of the egg (922). When rats were fed C^{14}-labeled malathion, the bulk (83%) of the activity was eliminated within 24 hours in the urine. A small amount of malathion was degraded to respiratory CO_2. Very little activity was found in the tissues or blood (1628).

Apparently two major degradative pathways occur in mammals: that involving carboxy esterases and that involving phosphatases. The differences in the activity of these two systems, in mammals as against insects, helps account for the difference in toxicity. In the urine of cows, the principal metabolite was malathion monoacid; the diacid became more important with time. Desmethyl malathion was also found. In fecal matter, malaoxon, dimethyl phosphate, and 0,0-dimethyl phosphorothioate were found (923, 1093, 1132, 1475, 1485). Other studies with rats and dogs have shown the presence of malaoxon in the blood and malathion mono- and di-acids in the urine as well as monomethyl phosphate, dimethyl phosphate, and dimethyl thiophosphate (807, 945, 1283).

The hydrolytic effect of various esterases on malathion was investigated. It was found that malathion was not hydrolyzed at a significant rate by horse-serum-cholinesterase, nor by pancreatic lipase. Rat liver aliesterase, however, hydrolyzed malathion to the monoacidic malathion. It was not certain which of the two possible acids was formed or whether both were formed (921).

In human liver, malathion was degraded mainly through the action of a carboxyesterase. Other hydrolytic actions occurred but at a rather low level: Demethylation accounted for about 5%; P-S cleavage, for about 1%; and S-C cleavage for about 0.1 to 0.5% (944).

Although both phosphatase and carboxyesterase activity are present in insects, it is the lower activity of the carboxyesterases and the higher production of malaoxon that apparently accounts for the higher insect toxicity (117, 300, 301, 303, 816, 817, 839, 868, 937, 938, 976, 1142, 1475). In flies resistant to malathion, the carboxyesterase activity was higher. Concomitant, there was significantly higher activity in degrading malathion to its monocarboxylic acid analog both *in vivo* and *in vitro* (941).

Studies have shown that enzyme systems are present in various tissues of desert locust, *Schistocerca gergaria* F., which can activate malathion. Activity was highest in fat bodies and gut and least in thoracic muscles, ovaries and head. S^{35}- and H^3-labeled malathion in mineral oil was applied to mature male locusts, *Schistocerca gregaria.* At varying intervals insect and excreta were analyzed. The authors felt that the data indicated activation of malathion and formation of malaoxon in the locust body wall (967).

After exposure of two-spotted spider mites to malathion, phosphate, diethyl malate, diethyl mercaptosuccinate, O,O-diethylphosphorothionate and malaoxon were detected (943).

The soil fungus, *Trichoderma viride,* apparently degraded malathion via two paths that did not include the oxidation product malaoxon. The presence of powerful carboxyesterases was suggested by the fact that the carboxylic acid derivatives of malathion constituted the major portion of the metabolites. Some variants of *T. viride* also showed high desmethylation activity. Diethylmalate and some unidentified hydrolysis products were also observed (935).

Malaoxon and three other products of malathion breakdown were recovered from lettuce sprayed with malathion (290). In other studies, dimethyldithiophosphate was found in plants treated with malathion (1415). When malathion treated maize and wheat was stored in sealed jars for six months and analyzed monthly, dimethyl phosphorothiolate, malathion monoacid, and malathion di-acid were identified by thin-layer chromatography. Other studies showed that wheat grain converted malathion to dimethyl phosphate and malaoxon. Malathion breakdown appeared, however, to be principally hydrolytic rather than oxidative (1244, 1245). Oxidation of the malathion to malaoxon occurred in the seed coats and germs of the wheat grains whereas the hydrolyses occurred in the germ and endosperm (1247).

In rice bran, the rate of disappearance of malathion was greater in those samples having the higher acid content. Hydrolysis proceeded, apparently, by both enzymatic and chemical routes. Dimethyl phosphorothionate and dimethyl phosphorothiolothionate were found (1249).

UV Irradiation of malathion dusts and emulsions showed the latter to be less stable (1104).

Activity of Carboxylesterase Toward Malathions

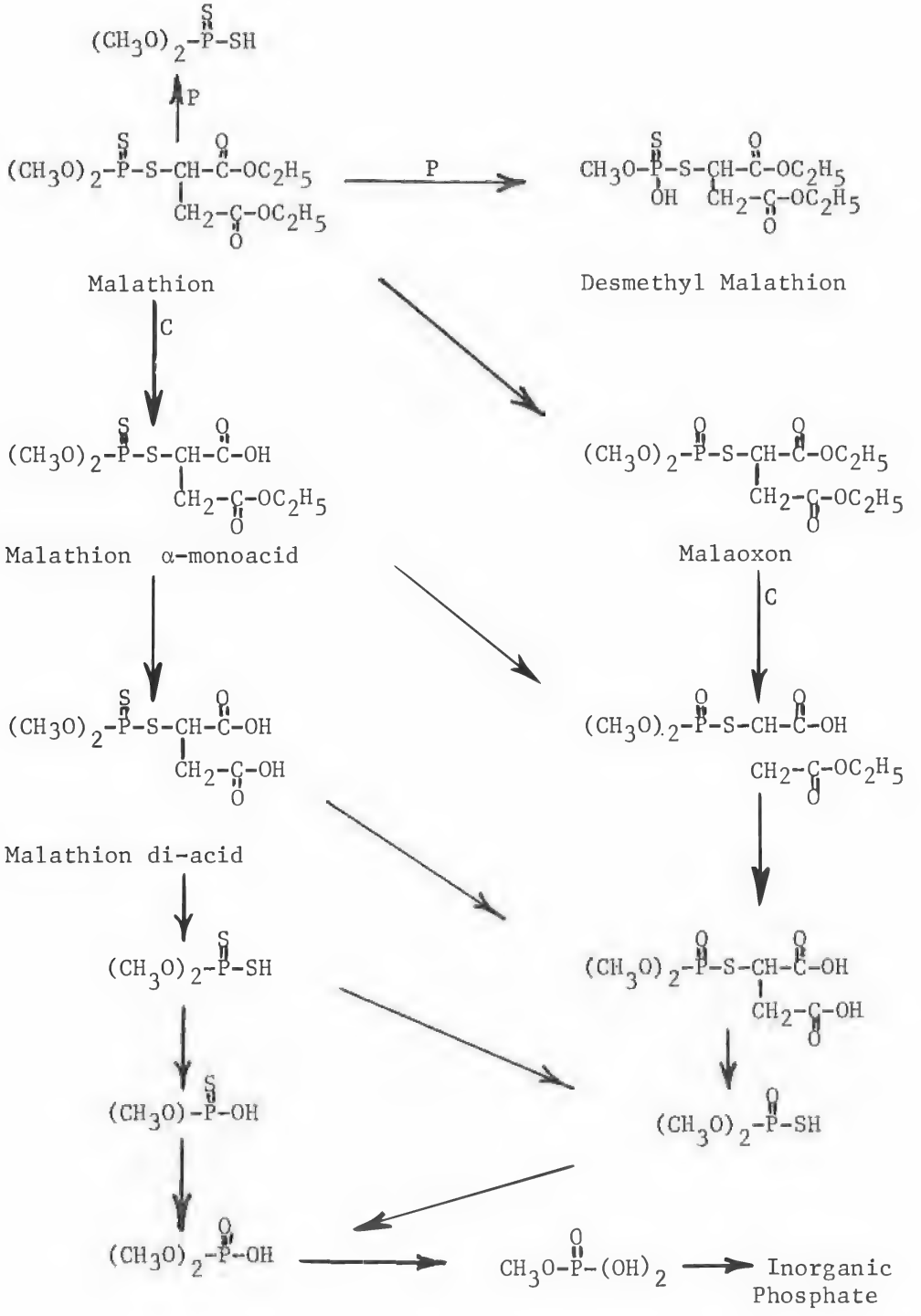
	K_m ± SE (mM)	V max ± SE μM/mg/min	Relative enzymatic half-life $0.695(K_m/V_m)$
d -Malathion	0.084 ± 0.007	0.761 ± 0.011	0.077
l -Malathion	0.159 ± 0.008	0.559 ± 0.008	0.218
dl-Malathion	0.205 ± 0.008	0.937 ± 0.043	0.152

Bimolecular Reaction Constants of Malaoxons

	$k_i \times 10^4 (M^{-1} min^{-1})$	
	Bovine erythrocyte AcCh esterase	Rat Liver carboxylesterase
d -Malaoxon	2.80	5.40
l -Malaoxon	0.63	0.65
dl-Malaoxon	1.49	1.70

(1673)

Studies with the optically active isomers of malathion and malaoxon showed that the d-isomer was more toxic that the l-isomer (1673).


P
P
Malathion
Desmethyl Malathion
C
Malathion α-monoacid
Malaoxon
C
Malathion di-acid
Inorganic Phosphate

Activity of Purified Rat Liver Carboxylesterase with Carbalkoxy Homologs of Malathion

	K_m ± SE (mM)	V max ± SE μM/mg/min	Relative enzymatic half-life $0.695(K_m/V_{max})$
Carbomethoxy	0.506 ± 0.075	0.52 ± 0.04	0.676
Carbethoxy	.179 ± 0.024	1.81 ± 0.12	.069
Carb-n-propoxy	.068 ± 0.018	2.46 ± 0.26	.019
Carb-i-propoxy	.040 ± 0.007	0.28 ± 0.02	.099
Carb-n-butoxy	.022 ± 0.005	2.97 ± 0.24	.005

Biomolecular Reaction Constants of Carbalkoxy Homologs of Malathion

	$K_i \times 10^4 (M^{-1} min^{-1})$		
	AcCH esterase	Serum cholinesterase	Liver carboxylesterase
Carbomethoxy	6.12	0.75	1.18
Carboethoxy	7.59	1.07	1.70
Carb-n-propoxy	1.78	11.2	8.26
Carb-i-propoxy	0.90	3.8	3.23
Carb-n-butoxy	1.78	32.3	37.4

(332)

	Bovine erythrocyte AcCh	
	Affinity =Ka($_m$M)	Phosphorylation Kp(min^{-1})
Malaoxon (dl)	3.6 ± 0.23	52.0 ± 2.1
Malaoxon (d)	2.3 ± 0.23	63.0 ± 3.2
Malaoxon (l)	4.9 ± 0.51	31.0 ± 2.3

(1788)

Using a rat liver carboxylesterase, the hydrolysis of malathion was investigated. NMR and infrared spectroscopy established that the monoacid was identical with the monoacid isolated from rat urine after oral administration of malathion. This was determined to be 0,0-dimethyl-S-(1-carboxy-2-carbethoxy)ethyl phosphorodithioate (α-monoacid) (1776).

Degradation rates of dusts at various temperatures were fitted to Jander's rate equation, when the rate of degradation did not exceed 80% (1389).

$$kt = [1-(1-\alpha)^{1/3}]^2$$

α = reaction rate

k = degradation rate constant

t = reaction time

T (°C)	k x 10^5
	Malathion dust
(Room temp.)	0.4
30	3.1
40	11
50	36
60	150

Maleic Hydrazide (MH) [6-hydroxy-3-(2H)pyridazinone]

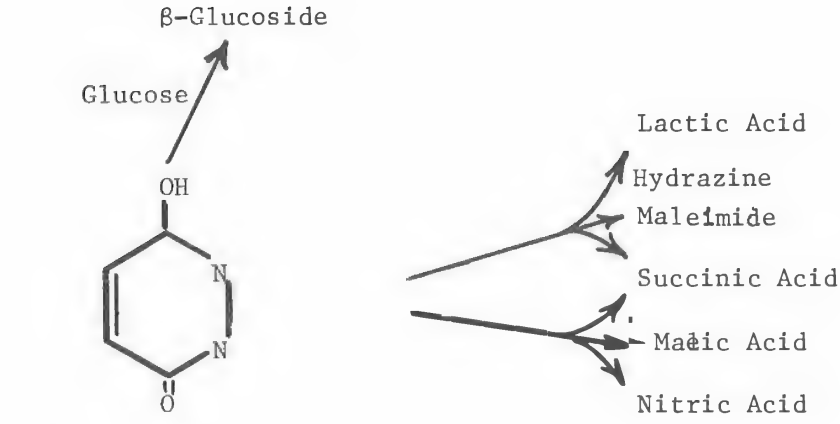
Ultraviolet studies indicated that maleic hydrazide exists as the hydroxy pyridazinone (1008).

C^{14}-MH was administered orally to rats. After three days, very little C^{14}-activity was detected in tissues or blood and CO_2 accounted for only 0.2% of the administered dose. MH was rapidly excreted via the urine unchanged (>90%) and as a conjugate (6-8%) (948).

When applied to tea, Camellia sinensis, MH was degraded to lactic acid, succinic acid, maleimide and hydrazine (1552). Extracts of treated wheat seedlings exhibited two spots on chromatograms, one of which was identified as a beta-glycoside of MH. In other studies with C^{14}-labeled maleic hydrazide and glucose, only β-glucoside was demonstrated (1420, 1484, 1672).

In the presence of oxygen, MH undergoes photolysis to form succinic, maleic and nitric acid; and in the absence of oxygen, to form succinic acid (1552).

Alcaligenes faecalis and Flavobacterium diffusum were isolated from sugar can soil in which the diethanolamine salt of MH was utilized (860).



Maleic Hydrazide

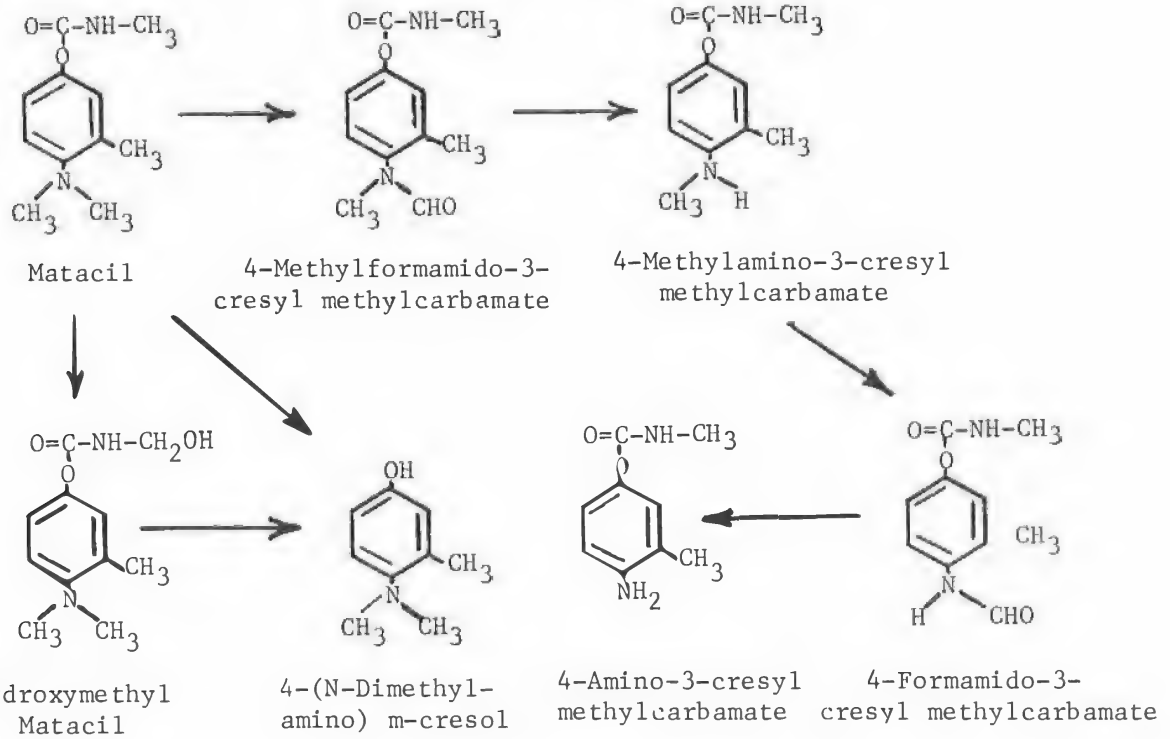
Matacil [4-Dimethylamino-3-cresyl N-methylcarbamate]

When labeled Matacil was administered to rats, $C^{14}O_2$ was observed. (836). After incubation with a rat liver microsome-reduced nicotinamide-adenine dinucleotide phosphate system, Matacil was degraded to N-hydroxy-methyl matacil, 4-amino-3-cresyl N-methylcarbamate, 4-methylamino-3-cresyl N-methylcarbamate (1110, 1111, 1112).

After exposure of flies to labeled Matacil, $C^{14}O_2$ arose through N-dealkylation and hydrolysis to the respective phenol and methylcarbamic acid. The latter decomposed to CO_2 via formate (1001). Incubation of matacil with fly homogenates and $NADPH_2$ gave rise to 4-formamido-, 4-methyl-formamido-, 4-amino-, and 4-methylamino-3- cresyl methylcarbamates (1427).

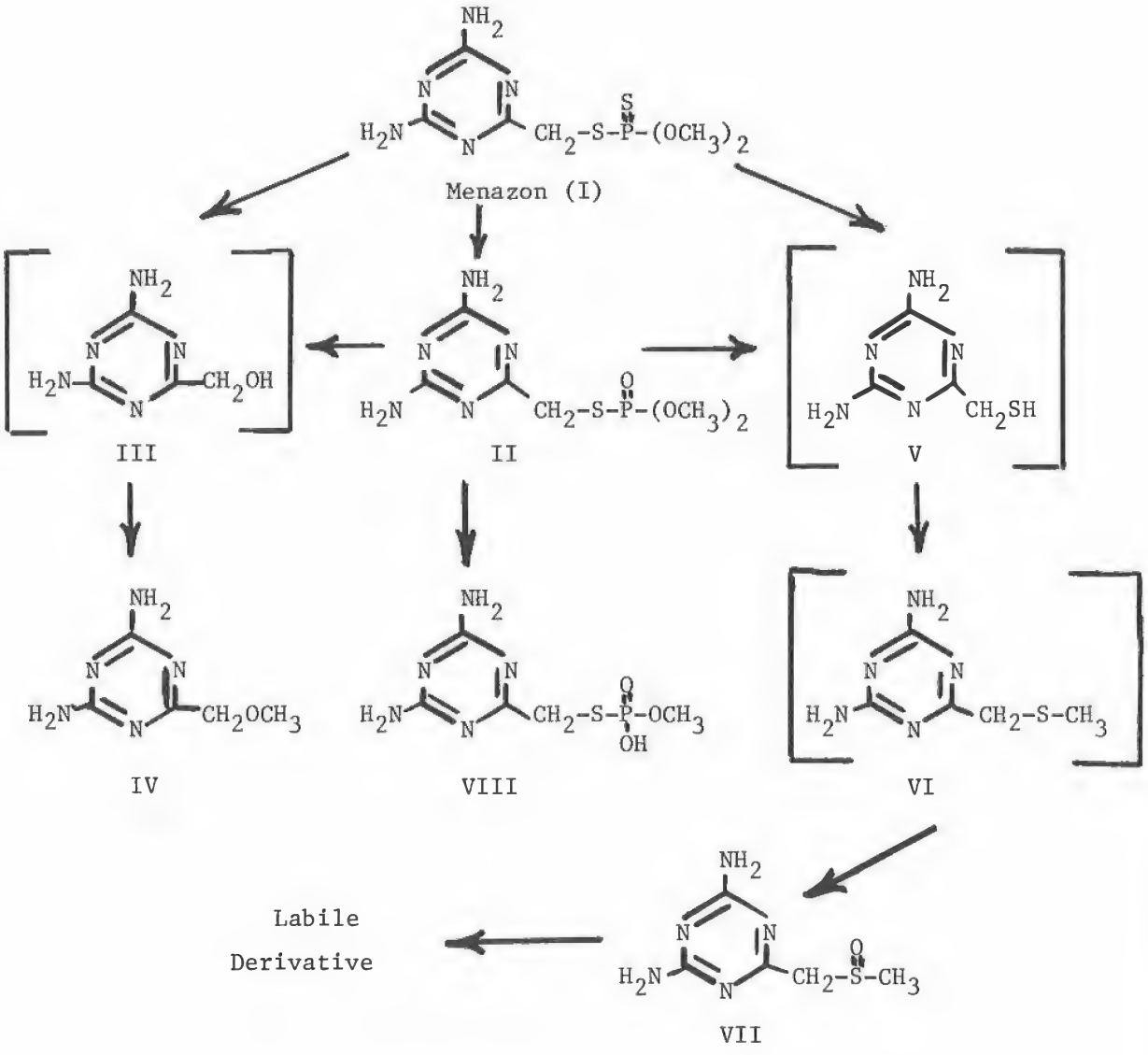
After treatment of growing bean leaves with labeled Matacil, at least eight compounds were detected. Of these, four were identified as 4-form-amido-, 4-methylformamido-, 4-amino-, and 4-methylamino-3-cresyl methyl-carbamates (2, 3, 845).

Exposure of Matacil to ultraviolet light (2537 A) produced at least eleven degradation products. Of these, the four compounds identified were identical with those found on bean plants (1, 2, 3).



Menazon [O,O-Dimethyl S-(4,6-diamino-s- triazin-2-ylmethyl) phosphorodithioate]

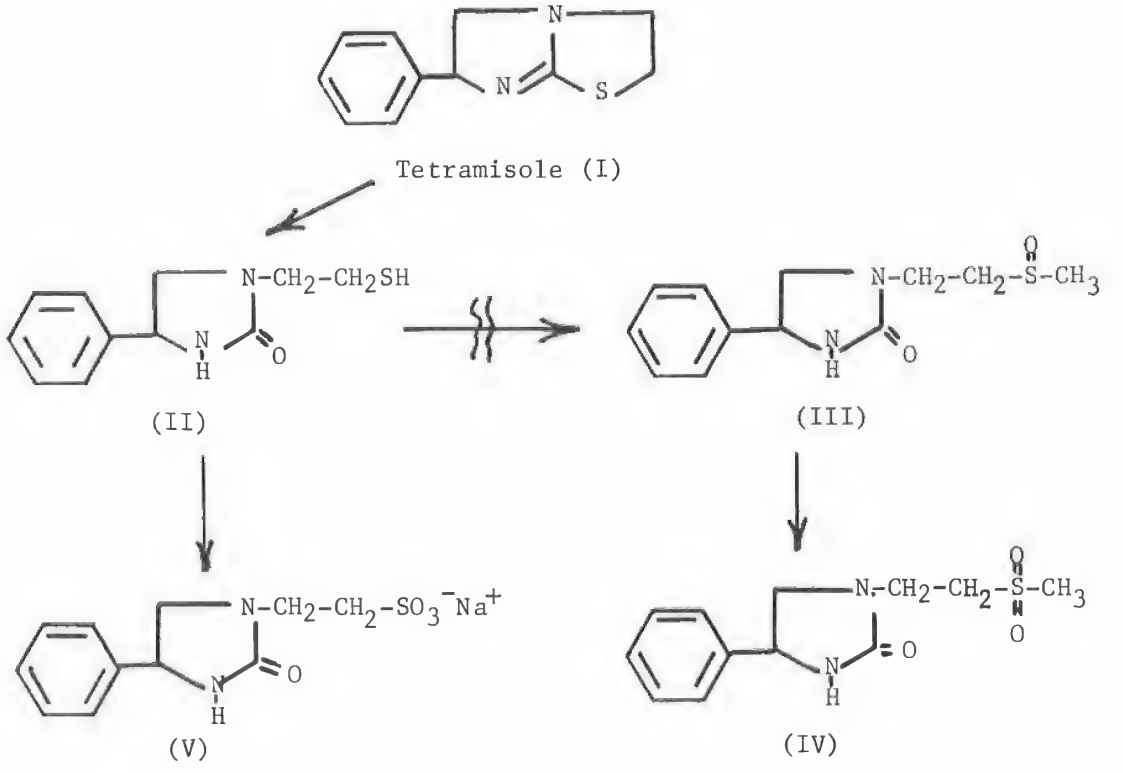
After an oral dose of C^{14}-labeled Menazon to rats, 80% of the label appeared in the urine within 24 hours. The major metabolites found in the urine were identified as compounds II, VII and VIII of the metabolic pathway shown. Metabolite IV and three unidentified compounds were also observed. It was felt that Menazon or its oxon analog was hydrolyzed and then methylated to give the methylthiotriazine (VI). This was then oxidized to the sulfoxide (VII) (519).



1-(2-Mercaptoethyl)-4-Phenyl-2-imidazolidinone

Forty-eight hours after oral administration of ^{14}C- and ^{35}S-labeled 1-(2-mercaptoethyl)-4-phenyl-2-imidazolidinone (II) to a rat, 66% of the ^{14}C and 76% of the ^{35}S radioactivity was excreted in the urine. Three compounds were identified: 1-[2-(methylsulfinyl)ethyl]-4-phenyl-2-imidazolidinone (III) (88%); 1-[2-(methylsulfonyl)ethyl]-4-phenyl-2-imidazolidinone (IV) (5%); and 1-sulfoethyl-4-phenyl-2-imidazolidinone (V) (4%) (1751).

Compound II was synthesized as a result of studies which indicated that it was a metabolite of 2,3,5,6-tetrahydro-6-phenylimidazo[2,1-b] thiazole, also known as tetramisole(I) (1751).



Mercury Compounds

$HgCl_2$ [Mercuric chloride]

Hg_2Cl_2 [Mercurous chloride]

The insecticidal action of mercury salts was related to their decomposition by soil organisms and the release of mercury vapors. This reaction required moisture and proceeded more rapidly as soil pH and temperature was increased (483). Mercury could be recovered from leaves of plants exposed to soil which had been moistened with bichloride of mercury (1543).

2-Chloro-4-hydroxymercuri phenol (I)

Cyano(Methylmercuri)guanidine (II)

Ethylmercury Acetate

Methylmercury dicyandiamide

Methylmercury chloride

Phenylmercury Acetate

Studies with organo-mercurials indicated that fungicidal inactivation occurred either by sorption, as with Trichoderma viride and 2-Chloro-4-hydroxymercuri phenol; or by sorption followed by metabolic breakdown of the fungicide as with compound Cyano(Methylmercuri)guanidine with Penicillium notatum or Aspergillus niger and compound Cyano(Methylmercuri)guanidine with Bacillus spp. (1338).

Non-volatile phenylmercury acetate was degraded to mercury and lost as a vapor. Ethylmercury acetate was also degraded to mercury but was lost as mercury and organomercury vapors. Methylmercury dicyandiamide and chloride did not give any significant metallic mercury vapor and their loss from soil was as the result of the volatility of the organo-mercurial (796).

Phenylmercuric acetate decomposed rapidly in a culture of wheat root tissue. Sulfhydryl groups accelerated decomposition (1572).

Dust formulations were illuminated with ultraviolet light (2537 A) and the rates of decomposition were determined (1387, 1388).

(1) $RHgOH + H^+ + e^- \rightarrow R\text{-}Hg\cdot + H_2O$

(2) $RHg\cdot + H^+ + e^- \rightarrow RH + Hg\downarrow$

		Mol x 10^{-4}	K(-Min)
PMC	Phenyl mercuric chloride	0.043	0.0251
EMC	Ethylmercuric chloride	8	0.0023
TMC	Tolylmercuric chloride	0.037	0.0151
PMP	Phenylmercuric propionate	10	0.0088
MEMC	Methoxyethylene mercuric chloride	20	0.0084
PMA	Phenyl mercuric acetate	20	0.0030

The effect of light on fungicidal activity in a series of compounds, EtHgX and Ph HgX (X=anion) was dependent on the nature of X: decreasing activity in the order, I >Br>Cl>OAc. In compounds RHgCl and $PhHgO_2CR$, resistance to the action of light increased as the carbon content of "R" increased. In $p\text{-}MeC_6H_4HgCl$, introduction of methyl groups meta to the original methyl group increased light resistance; other groups decreased resistance. Resistance was greater in the powder state than in solution or suspension. Fungicidal activity of ethylmercuric compounds decreased markedly after 52 hours of light exposure but that of phenylmercuric compounds did not (517).

Using arc lamps generating light of wavelengths longer than 2900A. The following compounds barely decomposed after the equivalent of 7 days' ultraviolet light: Phenylmercuric acetate, phenylmercuric chloride, phenylmercuric N-phenyl-p-toluene sulfamilide, and methylmercuric iodide. Activity of phenylmercuric dinaphthylmethane disulfonate fell markedly after 3 days and that of p-hydroxyphenylmercuric chloride fell slightly after 7 days (1291).

The partial pressure (P_t) of a mercurial molecular weight "M" may be expressed as

$$P_t = \frac{w}{M} \times 22.4 \times \frac{Tx760}{273.2}$$

where w = g/l mercurial in air at T° K.

$$\log_{10}P = a - \frac{b}{T}$$

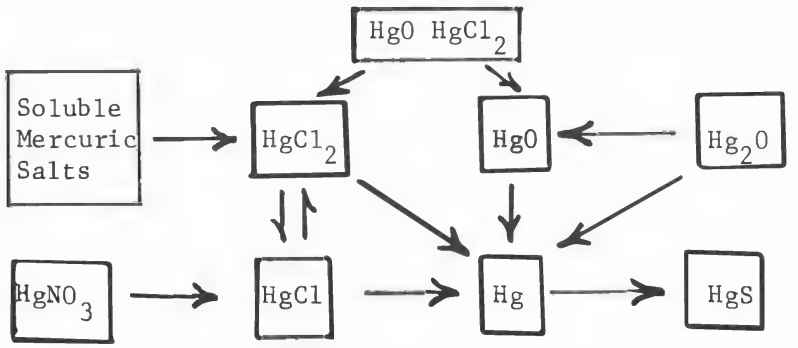
(1158)

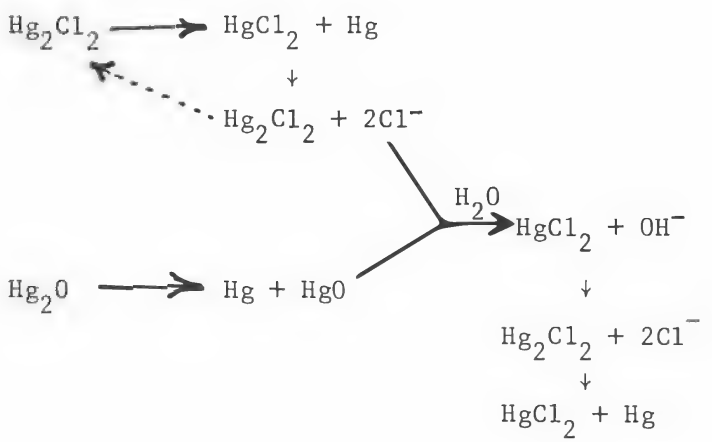
Constants in equation: $\log_{10}P = a - b/T$

Compound	a	b	Compound	a	b
1	11·14	3940	10	3·76	2380
2	8·64	3400	11	6·12	3175
3	8·04	3200	12	5·50	3140
4	9·26	3620	14	13·67	5755
5	10·11	3880	15	10·92	4800
6	10·49	4080	16	17·84	7050
7	12·22	4680	19	8·50	4300
8	9·20	3780			
9	10·67	4470			

1. Methylmercury chloride
2. Mercury (grey powder with talc)
3. Ethoxyethyl mercury silicate (tech.)
4. Methoxyethyl mercury silicate (tech.)
5. Ethylmercury chloride
6. Ethylmercury isothiourea hydrochloride
7. Methoxyethyl mercury chloride (tech.)
8. Ethoxyethyl mercury chloride (tech.)
9. Mercuric chloride
10. Ethylmercury dicyandiamide (tech).
11. Methylmercury dicyandiamide
12. bis-Ethylmercury phosphate
13. Tolylmercury acetate(mixed isomers?)
14. Phenylmercury acetate
15. Phenylmercury oxinate
16. Phenylmercury iso-urea
17. Phenylmercury salicylanilide (tech.)
18. Phenylmercury fluoroacetate
19. Phenylmercury chloride

The following scheme has been suggested to explain the behaviour of organomercurials in soil (139).





$$HgNO_3 \longrightarrow Hg + N_2 + O_2$$

$$\text{Phenyl-HgOAc} \rightarrow (\text{Phenyl})_2Hg + Hg + 2\ AcO^-$$

$$\text{Ethyl-HgOAc} \rightarrow (\text{Ethyl})_2Hg + Hg + 2\ AcO^-$$

$$CH_3O\text{-}C_2H_4\text{-HgOAc} \rightarrow CH_2O\text{-}C_2H_4\text{-}\underset{\downarrow}{Hg}\text{-Hg-}C_2H_4\text{-}OCH_3 + 2AcOH$$

$$Hg + HgOAc + CH_3OH + C_2H_4$$

Vapour pressure at 35° as function of substitution

(a) R·HgCl

R =	x 10^{-5} mm.
Me-	2300
Et-	300
$R'OC_2H_4$-	100
(R' = Me or Et)	
Ph-	0·4

(b) EtHg-X

X =	x 10^{-4} mm.
Chloride	30
isoThiourea	20
Dicyandiamide	1·1
Phosphate	0·2

(c) PhHg·X (static)

X =	x 10^{-6} mm.
Acetate	17
Oxinate	8
isoUrea	7
Fluoroacetate	6
Salicylanilide	5
Chloride	5
Methanodinaphthodisulphonate	2
Nitrate	1
Salicylate	1
Dithiocarbamate	0·8

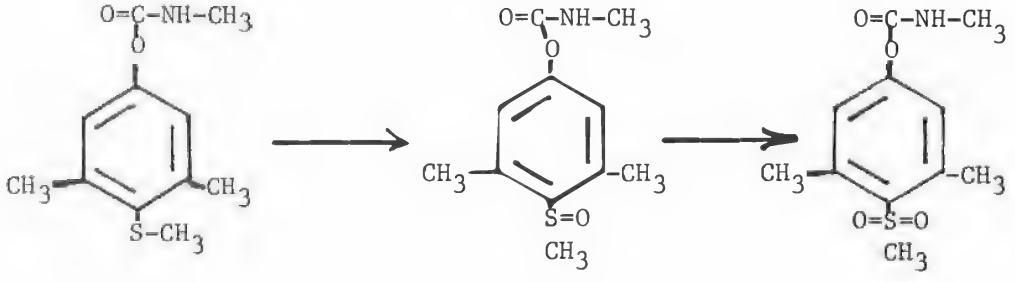
Mesurol [4-Methylthio-3,5-xylyl N-methylcarbamate]

Labeled Mesurol was degraded by rats with evolution of $C^{14}O_2$ (836). Mesurol was degraded by a rat liver microsome--$NADPH_2$ system to the sulfinyl and sulfonyl derivatives (1110, 1111, 1112).

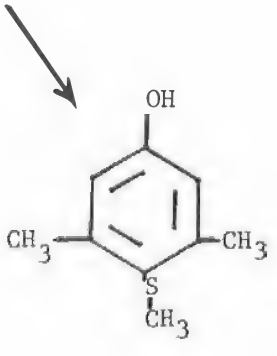
When flies were exposed to Mesurol, CO_2 arose through N-dealkylation and, after hydrolysis to the phenol and methylcarbamic acid, decomposition of methylcarbamic acid via formate (1001). After incubation of Mesurol with fly homogenate and $NADPH_2$, the sulfoxide was observed (1427).

On bean plants treated with Mesurol, both the sulfoxide and sulfone derivatives were identified (3, 845).

Exposure of mesurol to short-wavelength ultraviolet light (2537A) produced the sulfoxide, which in turn gave rise to three unidentified compounds, and the sulfone which also gave rise to two additional compounds (1, 3).



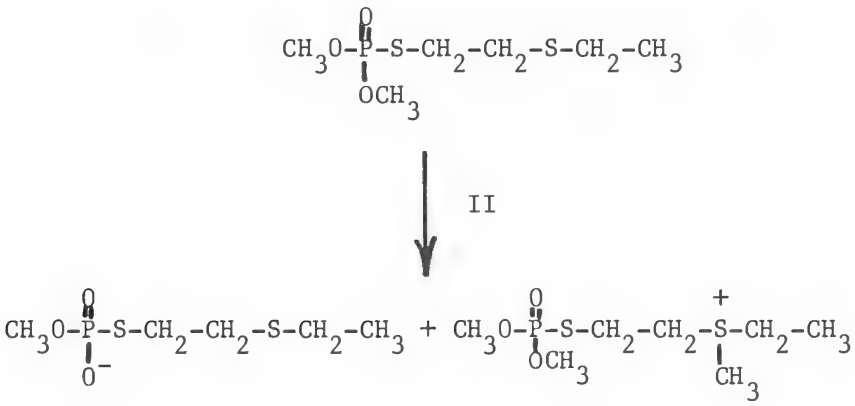
Mesurol



Metasystox - mixture of

0,0-Dimethyl O-ethylmercaptoethyl thiophosphate (I)
0,0-Dimethyl S-ethylmercaptoethyl phosphorothiolate (II)

Metabolism of metasystox is similar to that of systox. Some studies have shown that metasystox also undergoes a non-enzymatic self alkylation. There is some indication that systox may undergo the same reaction (1085).



Methoxychlor (1,1,1-Trichloro-2,2-bis-[p-methoxyphenyl]ethane)

Treatment of cows with methoxychlor apparently does not give rise to any residues in the milk. In other studies some metabolites have been isolated; however, none have been identified (285, 641, 859, 1179).

After prolonged incubation of methoxychlor with Aerobacter aerogenes, some of the olefin and the reductively dechlorinated pesticide were observed (974).

Methoxychlor was irradiated with high intensity ultraviolet energy between2200 and 3300 A. Dechlorination and cleavage occurred. Those compounds identified were: methoxyphenol; methylanisole; methoxychlor-DDE; 1,1,4,4-tetrakis(p-methoxyphenyl)-2,3-dichloro-2-butene; 1,1,4,4-tetrakis (p-methoxyphenyl)-1,2,3-butatriene; p,p'-dimethoxybenzophenone (1762).

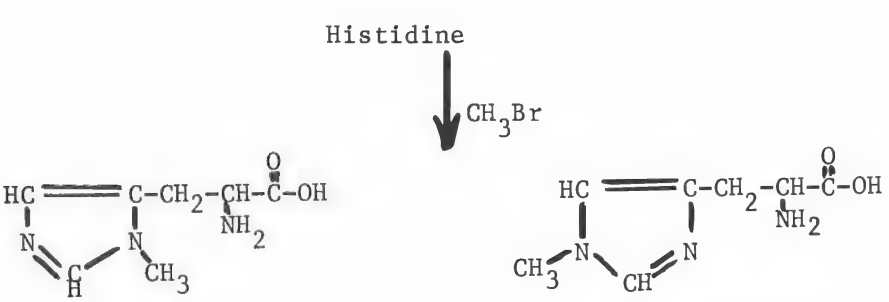
2-Methoxy-3,6-Dichlorobenzoic Acid

This compound is rapidly metabolized by wheat and bluegrass. One metabolite has been identified as 3,6-dichlorosalicylic acid. The other metabolite, representing 90% of the herbicide and believed to be a hydroxylated compound, has not been identified (1029).

Methyl Bromide

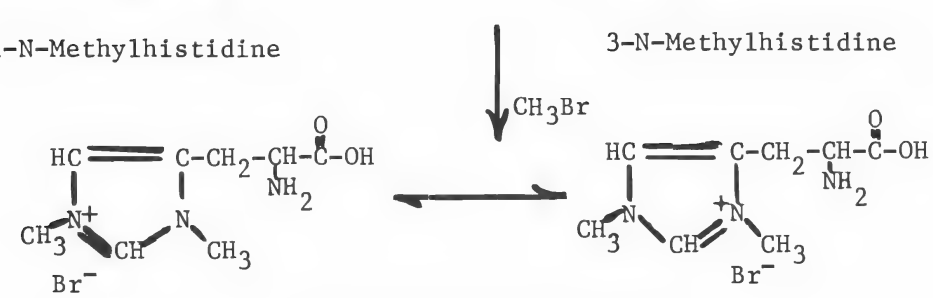
Under conditions of fumigation, methyl bromide absorbed by wheat underwent decomposition with the formation of inorganic bromide. The gluten or protein fraction of wheat accounted for most of the decomposition; 40% as N-methyl derivatives; 24% as dimethyl sulfonium derivatives; and 16% as methoxyl and thiomethoxyl derivatives. Some free methanol (10%) was produced by hydrolysis. Work on the methylation of wheat protein by methyl bromide has led to the isolation and identification of 1-N-methyl-histidine, 3-N-methylhistidine, 1,3-N,N'-dimethylhistidine, and ε-N-methyl lysine (175, 1516).

Histidine

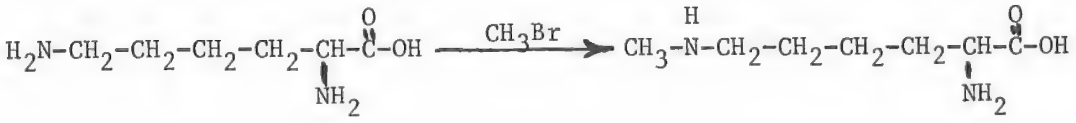


1-N-Methylhistidine

3-N-Methylhistidine



1,3-N,N'-Dimethylhistidine

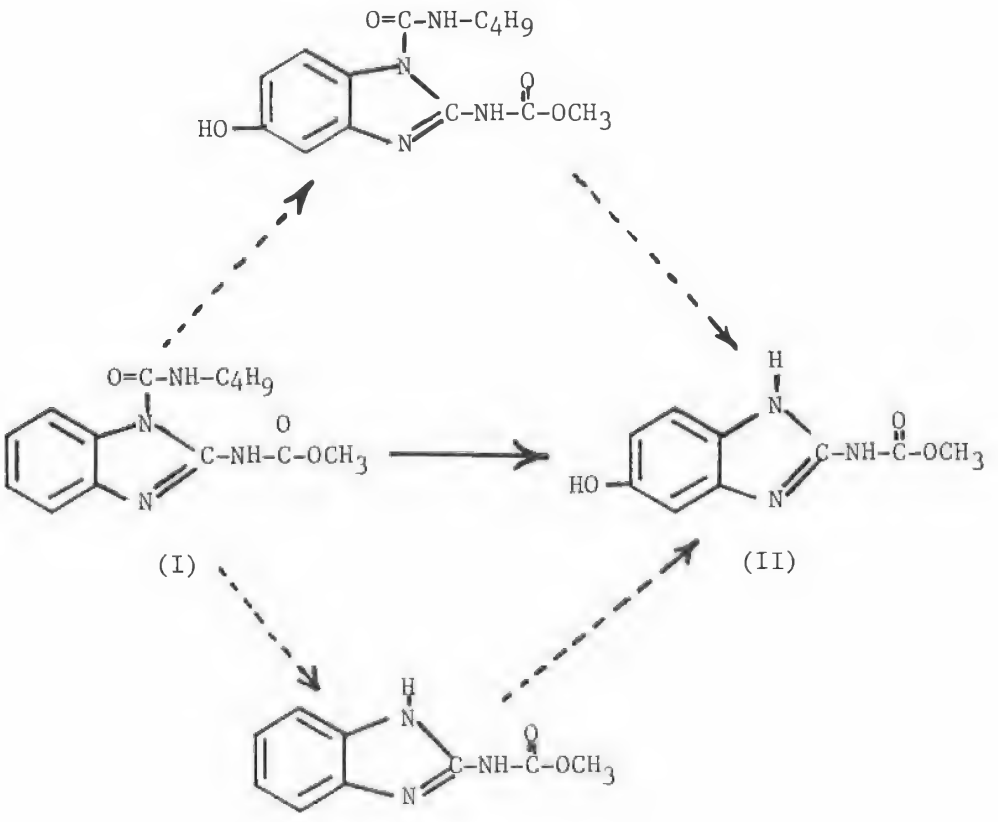


Lysine

ε-N-Methyl lysine

Methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate (DuPont Fungicide 1991)

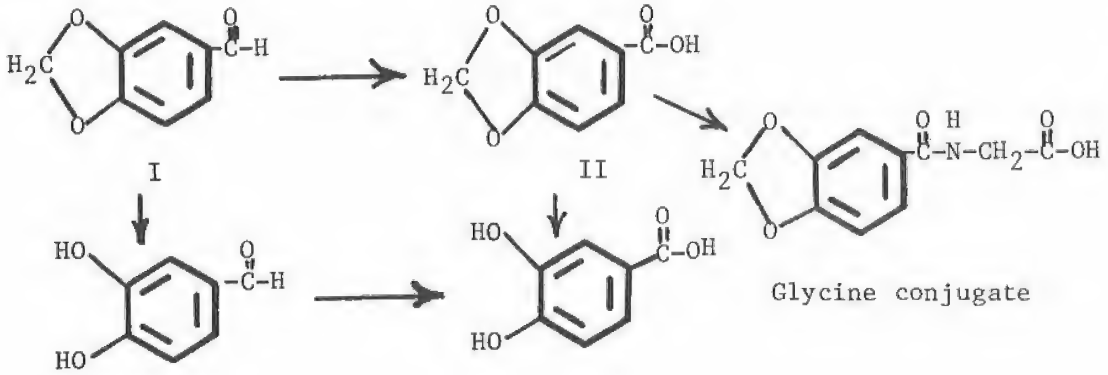
Analysis of composite urine samples from rats fed DuPont fungicide 1991 (I) was performed. Methyl 5-hydroxy-2-benzimidazolecarbamate (II) was found after enzyme hydrolysis to liberate glucuronide and/or sulfate conjugates. It was not determined whether hydroxylation occurred prior to or after loss of the butylcarbamoyl group (1733).



Methylenedioxyphenyl Compounds

Piperonal

Piperonal (I) was irradiated for periods of 1 - 24 hours when dry with a G.E. sunlamp (275W, peak absorbance 2967A). The exposure temperature was 53°C. Using gas and paper chromatography, three compounds were resolved and identified: piperonylic acid (II), protocatechualdehyde (III) and protocatechuic acid (IV). A dark brown product was also obtained from the reaction mixture. This was probably of a polymeric nature (458). When piperonal was fed to mice, piperonylic acid, N-piperonylglycine and two unidentified compounds were observed (755).



Piperonyl Alcohol
Piperonylic Acid

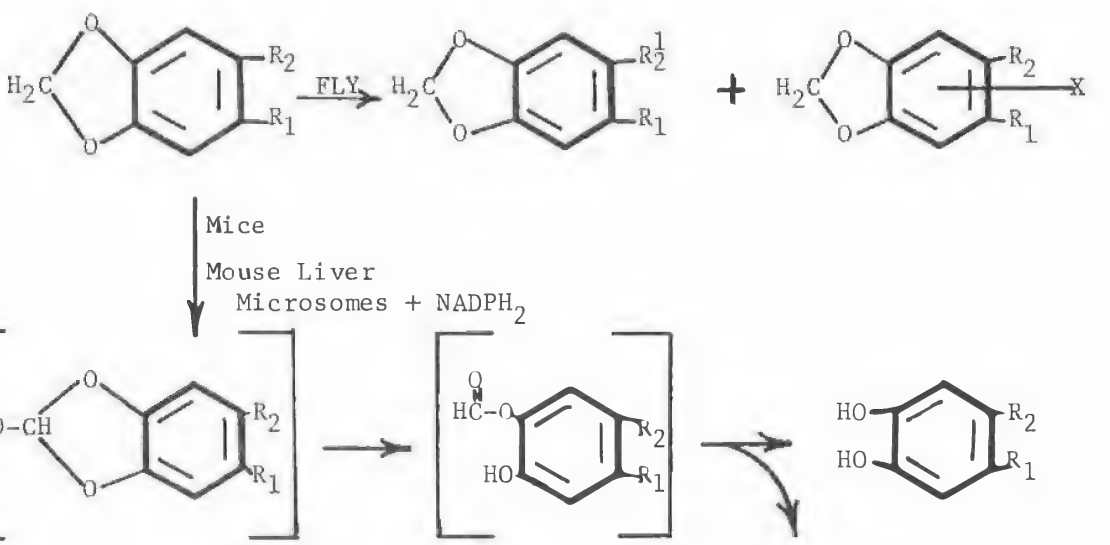
Piperonylic acid was found after feeding the alcohol to mice. Free acid and the glycine conjugate were found after feeding either compound (755).

Piperonyl Butoxide

Sulfoxide

Exposure of Madeira roaches to C^{14}-labeled piperonyl butoxide gave rise to several water soluble metabolites which were unidentified. Distribution of radioactivity showed that the brain and thoracic ganglia, fore- and hind-gut, and Malpighian tubules of the kidney contained the greatest amounts of radioactivity per unit weight (1275).

Within 48 hours after oral administration of methylene-C^{14} labeled dioxyphenyl compounds, mice excreted 61 to 76% of the label as $C^{14}O_2$. The amount excreted within 48 hours via urine varied with compounds. Metabolism of the methylenedioxyphenyl compounds in houseflies was apparently slower with less complete scission of methylenedioxy grouping than in mice. Piperonyl butoxide and sulfoxide gave rise to unidentified water soluble products. Mice degraded piperonyl butoxide to 8 compounds. The methylenedioxy moiety was cleaved and formate was produced (244, 755).



	R_2	R_1
Piperonal butoxide	$-CH_2-CH_2-CH_3$	$-CH_2-(OC_2H_4)_2-OC_4H_9$
Sulfoxide	$-H$	$-CH_2-CH(CH_3)-S(=O)-C_8H_{17}$

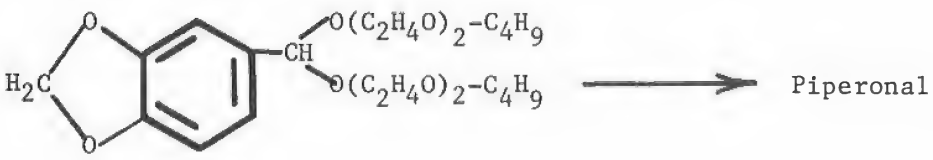
Tropital

When tropital was fed to mice, rats or hamsters, degradation occurred primarily at the side chain with excretion of metabolites in the urine. Some metabolism of the methylene dioxide to CO_2 also occurred. The glycine and glucuronate conjugates were found (755).

Rats, mice and rabbits, administered tropital orally, metabolized the tropital to piperonal, piperonylic acid, N-piperonylglycine and two unidentified acid compounds. When piperonylic acid was fed to mice, both the free acid and glycine conjugate were observed (755).

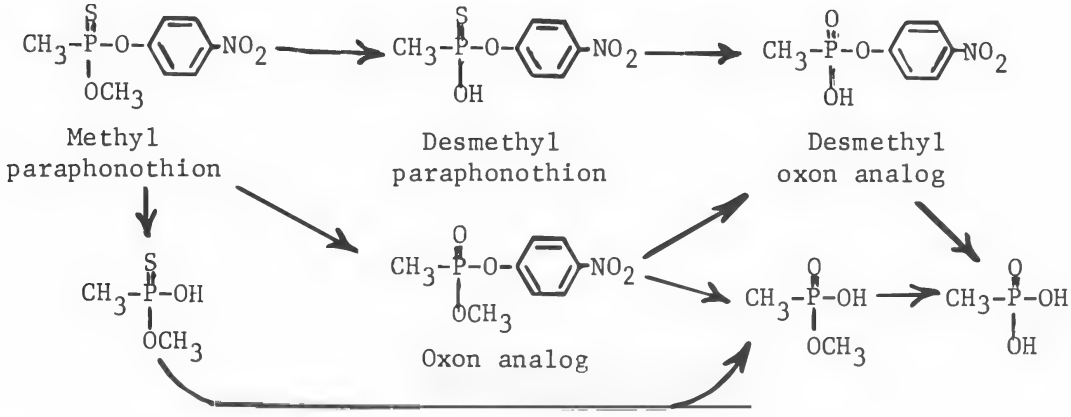
After incubation of tropital with mouse liver microsomes and $NADPH_2$, tropital was degraded to piperonylic acid via piperonal (755).

See also Piperonal.



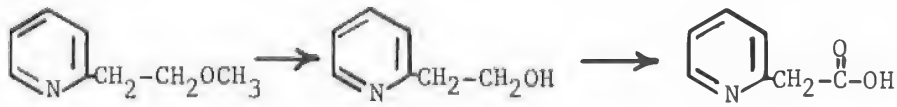
Methyl Paraphonothion [O-methyl O-p-nitrophenyl methylphosphonothionate]

P^{32}-labeled methyl paraphonothion was fed to Swiss mice. Urine and feces were collected and analyzed for excreted P^{32} compounds. There was no evidence of degradation of the P-CH_3 bond. All compounds were identified as hydrolysis products. Methyl phosphonic acid and its methy ester were present in the highest proportions at the three dosage levels tested. Additionally, O-methyl methylphosphonothioate, desmethyl methyl paraphonothion, its oxon analog, the desmethyl oxon analog, and two unidentified compounds were observed (682).



Methyridine [2-(2-methoxyethyl)-pyridine]

Methyridine and its metabolites were rapidly excreted in urine of sheep, calves, and small laboratory animals, irrespective of the route of dosing. Studies with labeled methyridine showed the presence of seven metabolites. Of these pyrid-2-yl ethanol, pyrid-2-yl acetic acid, and pyrid-2-yl acetylglycine were identified. Using liver homogenates, formaldehyde was also observed. There were indications that the pyridine nucleus had been altered in two of the metabolites (208).



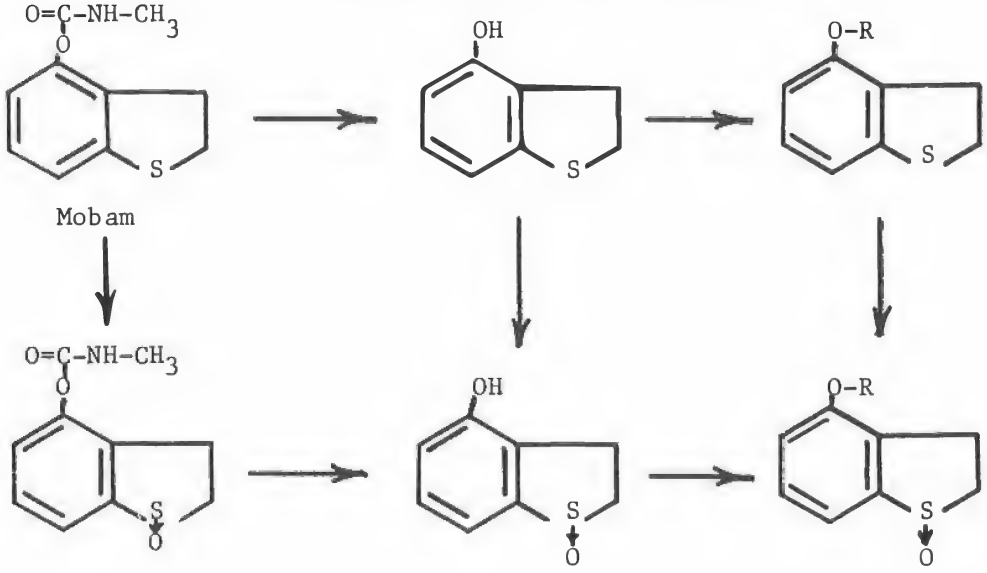
Methyridine

Mobam [4-Benzothienyl N-methylcarbamate]

In rats dosed with C^{14}-ring labeled Mobam, 76 - 87% of the label was excreted in 24 hours in urine. 4-Benzenethienyl sulfate and glucuronide accounted for 83 to 87% of excreted C^{14}. Metabolites were identified by infra-red (1216).

Within twenty-four hours after administration of ^{14}C-labeled Mobam to a dairy cow and a milking goat, 86% and 95% of the label had been excreted in the urine by the cow and goat, respectively. Approximately 12 and 5%, respectively, had been excreted in the feces. In the collected urines, after administration of methyl and carbonyl labeled Mobam, the sulfoxide, sulfoxide-4-sulfate, 4-sulfate and 4-glucuronide were identified. Seventy-five per cent of the label was collected within 24 hours as $^{14}CO_2$ (1756).

R= Glucuronide and/or sulfate



Mylone (3,5-dimethyl tetrahydro-1,3,5,2H thiadiazine-2-thione)

One hour after oral administration to rats, unhydrolyzed mylone was found in the stomach. Carbon disulfide equivalent to 1-3% of the dose was found in the stomach and 1.5% of the dose was found in exhaled air. After either intravenous or oral administration of mylone to dogs, CS_2 was found in the exhaled air of dogs (1334).

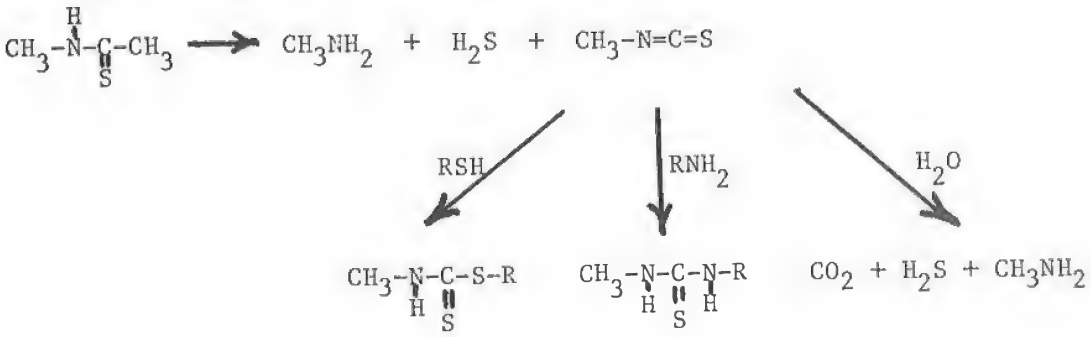
Activation or degradation of mylone in soil was dependent on moisture or free water and was a function of time and temperature. Formaldehyde was the first breakdown product and appeared within 15 minutes at room temperature. Methylamino methyldithiocarbamate appeared next and in turn formed CH_3NH_2, methyl isothiocyanate, and hydrogen sulfide. Monomethyl amine and hydrogen sulfide reacted with formaldehyde to form methyl amino methanol, dimethyl amino methane, and 1,3,5-trithiocyclohexane. Eventually this proceeded to CO_2, NH_3, SO_2, and H_2O. The methyl isothiocyanate reacted with H_2O to yield CO_2, H_2S, and CH_3NH_2 (1418). Methyl isothiocyanate may also react with ammonia, amines or sulfhydryls if present (960, 1052, 1054, 1418).

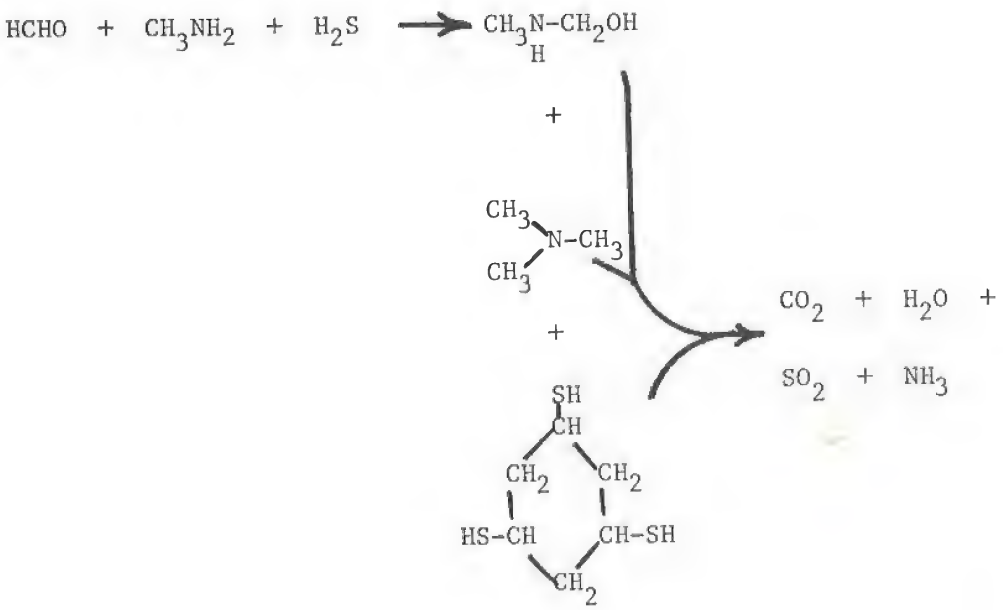
Studies indicated that clays--attapulgite, kaolinite, bentonite--act catalytically in the initial breakdown of mylone and facilitate the release of methylisothiocyanate (1055).

Mylone was not hydrolyzed within 7 days to an appreciable degree in water at 30°C. Increased temperature or the presence of acid increased hydrolysis and yielded 1 of carbon disulfide, two of formaldehyde, and two of methylamine per molecule of mylone (1334).

Methyl isothiocyanate release was found to be a first order reaction and varied directly with temperature and moisture; increased with soil pH over the range of 2.3 to 6.5 but fell off at values up to pH 7.7; decreased with increased clay or peat content; but was not dependent upon soil organisms (1052).

Mylone





Naphthalene Acetic Acid (NAA)

Naphthalene Acetamide (NAD)

After single oral doses of naphthaleneacetate in rats, four metabolites were found in the urine. Two were identified as naphthaceturic acid and naphthacetylglucosiduronic acid. A third compound was apparently as a sulfate, indicating possible hydroxylation. The fourth was unidentified (1695). Hydrolysis of the methyl ester of naphthaleneacetic acid was observed during storage of treated potatoes (1546).

When Winesap and Stayman cultivars were exposed to NAA or NAD, some decarboxylation occurred and six conjugates were formed. Pea roots exposed to NAA produced NAA-aspartate (1548).

Exposure of NAA to ultraviolet light and sunlight produced 1-hydroxymethyl naphthalene, 1-naphthaldehyde, naphthalene-1-carboxylic acid and 1-methyl naphthalene under aerobic conditions. Anaerobically, only the 1-methyl analog was observed (1391) Phthalic acid has also been observed (1791).

When NAA was incubated with Aspergillus niger , the major metabolite was found to be the 5-hydroxy analog. The 4- and 6-hydroxy analogs were also observed(1731).

Nellite [Phenyl-N,N'-dimethylphosphorodiamidate]

Metabolism studies were carried out with 1-month-old soil grown cucumber plants (Cucumis sativus). When phenyl-1-^{14}C-labeled Nellite was used, phenol was isolated and identified. Hydrogen phenyl methylphosphoramidate and dihydrogen phenyl phosphate were provisionally identified in the same fraction. From another fraction, which contained lipophilic materials, phenol and dihydrogen phenyl phosphate were obtained after alkaline hydrolysis (969, 1740).

Nicotine [1-methyl-2-(3-pyridyl)pyrrolidine]

In man, nicotine is metabolized to cotinine, hydroxycotinine, desmethylcotinine, and other unidentified compounds (148, 149). In other studies with C^{14}-labeled nicotine, the nicotine isomethonium ion was isolated from urine after intravenous administration (956).

After administration of nicotine to dogs, both orally and intravenously, urine was collected and analyzed. There was no prolonged storage of nicotine since about 95% appeared in the urine after 36 hours (106). A number of compounds were isolated from the urine and identified: (-) cotinine (III), (-) desmethylcotinine (V), hydroxycotinine (VI), γ-methylamino-γ-(2-pyridyl) butyric acid (II), β-oxo-γ-(3-pyridyl)-N-methylbutyramide (IV), 3-pyridylacetic acid (VII). A variety of other compounds resulted from demethylation of cotinine and production of one-carbon metabolites and from the loss of two labeled carbons in the pyrrolidine ring. Tests also showed that V spontaneously cyclized to the lactam (III) at physiological pH (854, 953, 954, 955, 956, 958). The nicotine isomethonium ion was also found in urine of dogs to whom nicotine had been administered (1431).

After administration of cotinine to dogs, (-) desmethylcotinine (V), hydroxycotinine (VI) and a compound thought to be butyramide (IV) were isolated from urine (952, 957).

In vitro studies with rabbit liver microsomes, TPNH, and O_2, indicated that metabolism of nicotine proceeded through hydroxylation to 5-(3'pyridyl)-N-methylpyrrolidine-2-ol; oxidation to cotinine; and deamidation of cotinine to 4-(3'pyridyl)-4-methylamino-butyric acid. No CO_2 was observed (696, 699, 700).

After application of nicotine to mustard green, cotinine was shown to be the principal metabolite (577).

Studies indicated that cotinine was the principal metabolite with insects also. However, the number of metabolites found varied; with tobacco wireworm, 1; with cigarette beetle, 2; with differential grasshopper, 4; and with the housefly, 3 (1281). In German and American cockroaches, too, cotinine was the main metabolite. In the southern armyworm, a number of metabolites were found but not identified (592).

The initial step in the bacterial decomposition of nicotine appeared to be conversion to N-methyl-3-(2 -pyrryl)pyridinium hydroxide (275).

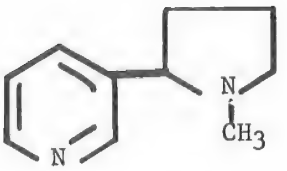
The first product of nicotine oxidation by Arthrobacter oxydans, a soil microorganism, was identified as (1)-6-hydroxynicotine. In subsequent studies, 6-hydroxypseudooxynicotine was identified and 6-hydroxy-N-methylmyosmine was postulated as the intermediate (671, 672, 673).

Decomposition of nicotine by pseudomonas gave rise to oxynicotine, pseudooxynicotine, and 3-nicotinoylpropionic acid (1458). In other studies, γ-(3-pyridyl)- -methylaminobutyric acid was believed to be an intermediate in the biological degradation of nicotine (955).

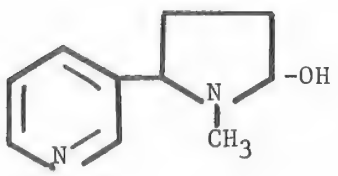
Nicotine was incubated with Ps. nicotinopbaga. In addition to pseudooxynicotine, 3-succinoyl pyridine, 3-succinoyl pyridone-6, and methyl amine were also observed. The process of degradation by bacteria was indicated as proceeding initially through N-methylmyosmine with direct decomposition of the pyridone nucleus (1384, 1385).

Other sutides have shown that nicotine was degraded by B. nicotianum, B. nicotinophagum and B. nicotinovorum. Among the degradation products found was N-methylmyosmine. A soil bacterium, probably Pseudomonas, converted nicotine to several compounds identified as pseudo oxynicotine, 3-pyridyl-3-methyl-aminopropyl ketone, and a compound believed to be 3-succinoyl pyridine. Decomposition products of the latter were not identified (195, 196, 1457, 1486, 1487).

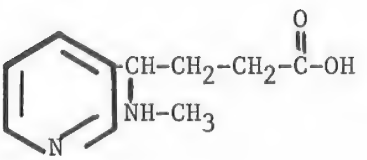
Nicotine decomposition was accelerated by ultraviolet radiation. The rate was greater in solution at pH 9.5 than at acid pH of 6.2 or 2.1. The sulfate form was more stable than free nicotine (1103). Autoxidation of nicotine gave rise to cotinine (481).



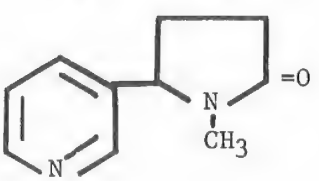
Nicotine (I)



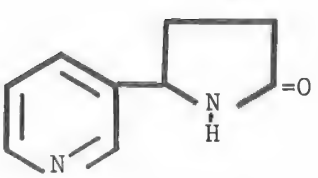
5-(3'-pyridyl)-N-methyl-pyrrolidine-2-ol (VIII)



γ-methylamino-γ-(3-pyridyl) butyric acid (II)

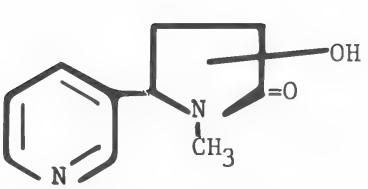


Cotinine (III)

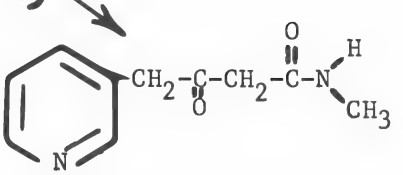


Desmethylcotinine (V)

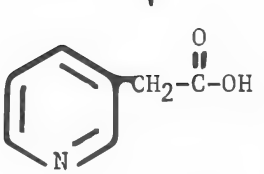
+



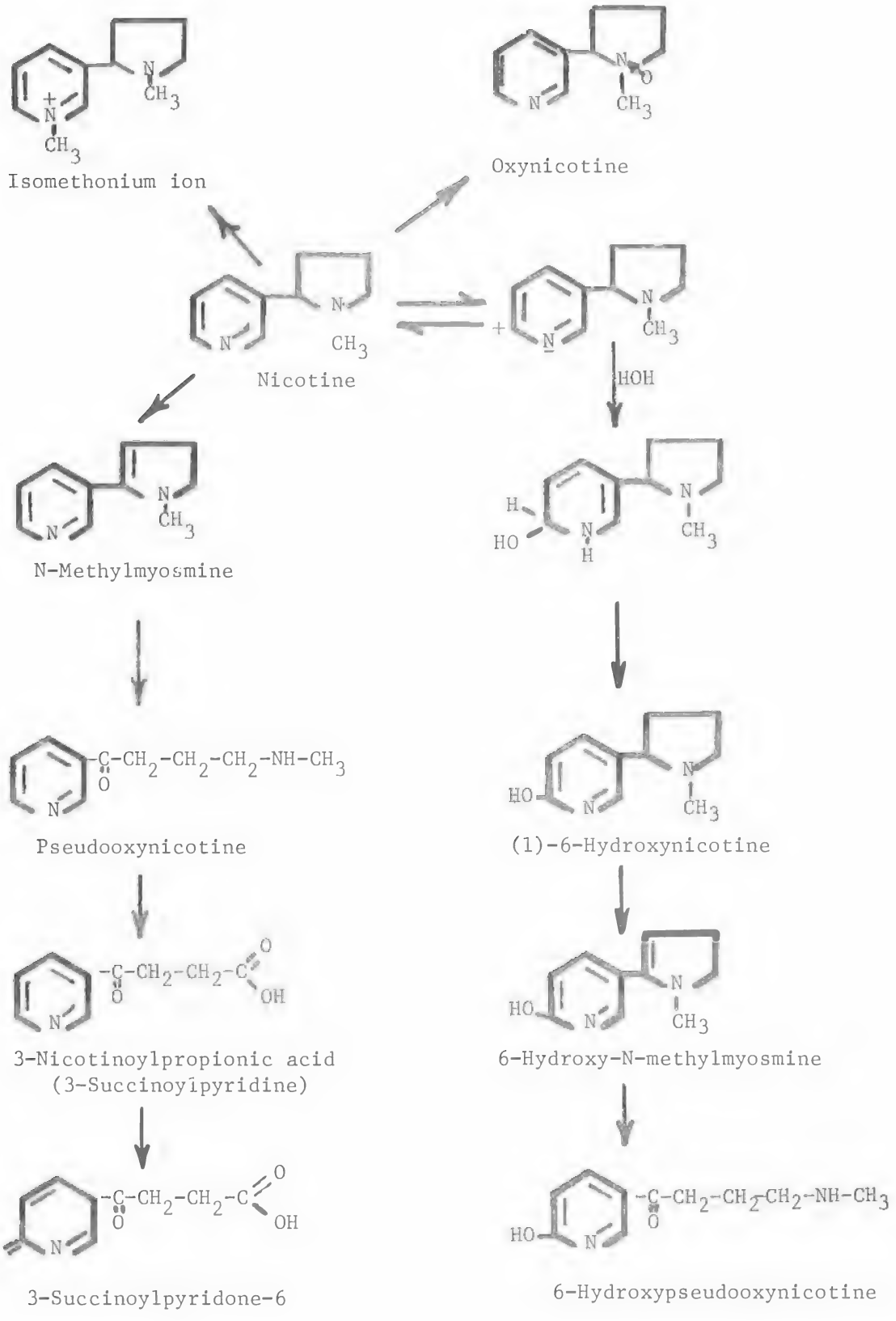
Hydroxycotinine (VI)



β-oxo-γ-(3-pyridyl)-N-methyl-butyramide (IV)



3-Pyridylacetic acid (VII)


Isomethonium ion
Oxynicotine
Nicotine
HOH
N-Methylmyosmine
Pseudooxynicotine
(1)-6-Hydroxynicotine
3-Nicotinoylpropionic acid
(3-Succinoylpyridine)
6-Hydroxy-N-methylmyosmine
3-Succinoylpyridone-6
6-Hydroxypseudooxynicotine

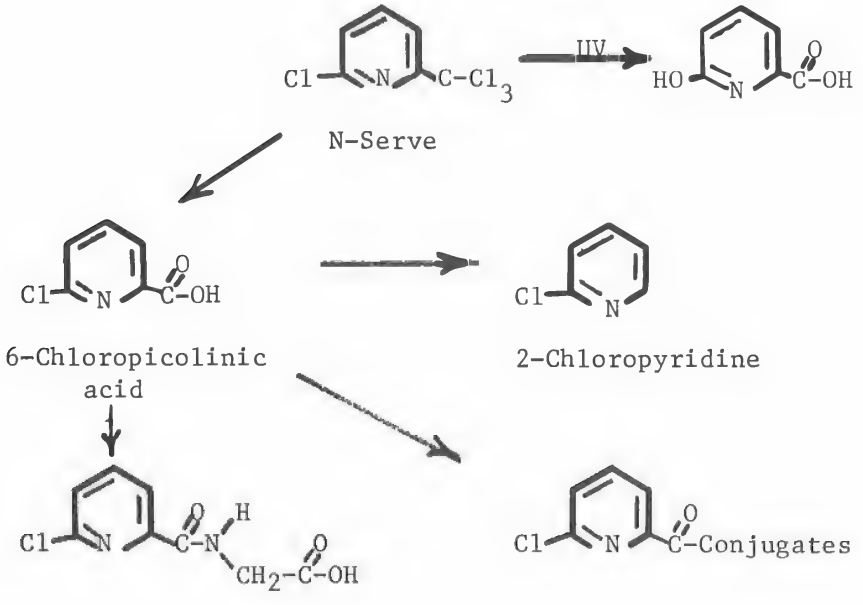
N-Serve
[2-Chloro-6-(trichloromethyl)pyridine]

C^{14}-labeled N-Serve was fed to a female beagle. Paper chromatography of the collected urine was used to isolate metabolites. Mixed melting points, UV, paper chromatography, and infrared spectra were used to identify the metabolite as N-(6-chloropicolinoyl)glycine (1202, 1203).

After the ingestion of N-Serve, rats excreted via the urine 6-chloropicolinic acid (mainly) and N-(6-chloropicolinoyl)glycine (1198).

Studies were conducted with labeled N-Serve and corn, carrots, lettuce, tomatoes, wheat and oats. Fractionation and chromatography showed that the principal metabolite was 6-chloropicolinic acid (1198, 1199, 1200). Corn converted the 6-chloropicolinic acid to 2-chloropyridine. 6-Chloropicolinic acid was also conjugated with lipids and protein. Some free Cl^- was formed (970).

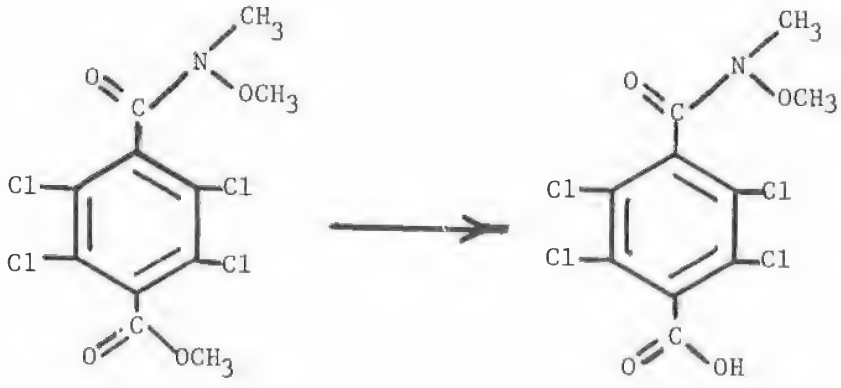
In soil, 2-chloro-6-(trichloromethyl) pyridine was hydrolyzed to 6-chloropicolinic acid. Samples of the acid were dissolved in excess 0.05N-$NaHCO_3$, poured in shallow glass dishes, and placed in direct sunlight, or irradiated with a mercury vapor lamp. 6-hydroxypicolinic acid was detected only in the mercury lamp irradiated solutions (1198, 1204).



OCS-21693 [Methyl 2,3,5,6-Tetrachloro-N-Methoxy-N-Methylterephthalamate]

After exposure of a Holstein cow to this compound, it was excreted as the hydrolysis product, 2,3,5,6-tetrachloro-4-carboxy-N-methoxy-N-methylbenzamide, in urine and feces. Similarly, the herbicide was rapidly hydrolyzed by beef liver slices or homogenate. When incubated with fresh rumen fluid for six hours, no decomposition was observed (1727).

(See also Dacthal)



OCS-21693

Parathion (E-605)
0,0-Diethyl 0-p-nitrophenyl phosphorothioate (Parathion)
0,0-Dimethyl 0-p-nitrophenyl phosphorothioate (Methyl Parathion)

Investigations have shown that the hydrolysis of parathion to paraoxon by liver was common to many animals: turtle (Pseudomys sp.), brook trout, brown trout, mud puppy, frog (Rana pipiens), pigeon, rabbit (Dutch belted), guinea pig, toad (Bufo sp.) mouse, rat and pig (Yorkshire) (1176).

Application of parathion and paraoxon to the skin of man, cat, rabbit, and rat showed that only the paraoxon was hydrolyzed (489, 491, 736). In man, the only metabolite identified was p-nitrophenol in urine (507, 873). More extensive studies with labeled parathion conducted with rats showed that highest levels of radioactivity appeared in the salivary glands and cervical brown fat. Liver, kidney, and adipose tissues also showed high radioactivity. Labeled material was excreted mainly by the kidneys and not in bile (490).

Other studies with rats have shown that parathion metabolism proceeded via de-alkylation as well as paraoxon. Mono- and di-methyl phosphate, dimethyl phosphorothioate, thiophosphate, inorganic phosphate and p-nitrophenol were also observed (13, 181, 350, 369, 499, 500, 538, 843, 1016, 1017, 1021, 1022, 1063, 1443). Similar results were obtained when methyl parathion was fed to male Swiss mice (682).

When parathion was incubated with bovine rumen fluid, paraoxon, amino parathion, and amino paraoxon were produced along with diethyl phosphoric and phosphorothioic acids (10, 299, 1125). In the case of nonruminating mammals, p-nitrophenol was excreted in the urine (401, 526). In cattle, however, conjugated p-aminophenol was excreted in the urine, probably the result of the ability of rumen organisms to reduce parathion to amino parathion.

Studies have also shown that mammalian blood was capable of hydrolyzing parathion (412, 1078).

Liver homogenates from several species of mammals, birds and fish were used to study nitroreductase activity. A comparison of the data showed that enzymatic activity varied between classes and among species within a given class of animals. Although reduction of the nitro group to the amino group probably proceeded via the nitroso and hydroxylamino derivatives, the presence of neither one was detected. Species tested included: rat, mouse, guinea pig, chicken, English sparrows, bullhead, sucker, flounder, sculpin, large mouth bass, sunfish, bluegill, alewife. Distribution of nitroreductase in rats varied among tissues: liver> kidney> spleen> heart>

lung> erythrocytes> plasma =0. In English sparrows and chickens, activity in kidney exceeded that in liver (669).

The rate of degradation of parathion by rat and human liver was low (944). When rat liver microsomes metabolized parathion, the major metabolites identified were: desethyl paraoxon, paraoxon, diethyl phosphate, diethyl phosphorothionate and p-nitrophenol. Similar results were obtained with guinea pigs and rabbits (899, 1068, 1076, 1077, 1239, 1630). When methyl parathion was used, the desmethyl analog was observed (499).

During studies of the inhibition of trypsin and chymotrypsin, p-nitrophenol was released (624, 791). This material was capable of forming a complex with glutamic acid in the myosin molecule (844).

Parathion-S^{35} was applied to a cranberry bog. Flooding was done 24 hours after application. One liter of the water was added to an aquarium containing fresh water mussels (Elliptio companatus Solander) and estuarine fish (Fundulus heteroclitus L.). Three metabolites were detected in the fish. One was identified as aminoparathion. However, it was possible that the aminoparathion was formed by microorganisms rather than by the metabolism of the fish (1006).

Activation and degradation of methylparathion by liver microsomes of chickens, muscovey ducks, albino rats, mice guinea pigs, sheep, cattle, and hogs was studied. Percent activation of methyl parathion (see table II) was calculated from the following formula (737).

$$pI_{50} = \frac{I_{50} \text{ directly assay P(O) analogue}}{I_{50} \text{ incubated insecticide}} \times 10$$

After incubation of methyl, ethyl, propyl or isopropyl parathion with microsomes from houseflies, rat liver or rabbit liver, the corresponding phenol was found in each case. Each compound was also converted to its corresponding oxon analog. Some conversion of the oxons to phenols also occurred. These reactions occurred only in the presence of $NADPH_2 + O_2$ (1700).

Liver homogenates were fortified with NADP and incubated with methylparathion and its oxon analog. Decomposition products are tabulated (Table I) (1741).

Table I

Metabolite	Compounds Added: Methylparathion	Compounds Added: Methylparaoxon
Guinea Pig		
Oxon	3.55%	------
Desmethylphosphorothioate	9.65	------
Desmethylphosphorate	1.85	12.77%
4-nitrophenol	2.94	52.11
Unidentified	2.81	1.79
Rat		
Oxon	4.47	------
Desmethylphosphorothioate	12.19	------
Desmethylphosphorate	0.84	19.94
4-nitrophenol	3.00	9.37
Unidentified	0.76	2.20
Mouse		
Oxon	2.43	-----
Desmethylphosphorothioate	18.25	-----
Desmethylphosphorate	2.40	34.36
4-nitrophenol	3.55	6.24
Unidentified	0.26	0.30

Table II

Animal	Methyl parathion activation pI_{50} ♂	Methyl parathion activation pI_{50} ♀	Methyl paraoxon degradation pI_{50} ♂	Methyl paraoxon degradation pI_{50} ♀
Mouse	6.64±0.04	6.77±0.03	7.15±0.00	7.10±0.01
Rat	7.01± .10	5.68± .05	7.14± .01	7.37± .01
Guinea pig	5.52± .04	5.20± .07	6.96± .12	6.91± .00
Cattle	6.89± .09	6.10± .02	7.44± .07	7.43± .08
Hog	5.36± .03	5.66± .02	7.32± .02	7.16± .03
Sheep	5.73± .11	4.50± .01	7.23± .03	6.62± .10
Chicken	5.34± .06	6.17± .02	7.36± .08	7.36± .02
Duck	6.68± .02	7.07± .07	7.33± .01	7.22± .01

Studies with insects indicated paths of metabolism similar to that of mammals, via paraoxon and/or dealkylation, but at varying rates (500, 840, 891, 940, 995, 1022, 1096, 1166, 1299). In larvae of the rice stem borer, ethyl parathion, ethyl paraoxon, diethyl thiophosphate, and diethyl phosphate were found after topical application (818, 819).

From army worms (Mamestra brassicae, Noctuidae), three enzymatic fractions were obtained that were capable of hydrolyzing phosphate esters (722, 723). Other studies showed that the fat body of Periplaneta americana L. shared with mammalian liver the ability to convert parathion to paraoxon (820). When methylparathion was incubated with preparations from mid-gut of horn beetle larvae (Xylotrupes dichotomus L.), both the desmethyl analog of methylparathion and phosphoric acid were detected (501).

Female house flies were treated topically with P^{32} labeled methyl parathion. A total of nine metabolites were observed on thin-layer plates. In addition to an unidentified compound, orthophosphate, monomethyl and dimethyl phosphate, methyl paraoxon, phosphorothioate, dimethylphosphorothioate, desmethyl analog of methyl parathion and methyl paraoxon were observed (683).

A fine discussion of the influence of esterases in insects on the degradation of organophosphates of the E-605 series can also be found in Pflanzschutz-Nachrichten 19, 1-34 (1966).

When P^{32}-labeled methyl parathion was applied to cotton leaves, analyses indicated a half-life of 24 hours. Four compounds containing P^{32} were found in the residue. Two were identified as methyl parathion and methyl paraoxon and two were unidentified. Under conditions of high temperatures more of the methyl parathion in the cotton leaf was converted to methyl paraoxon. Other studies with plants have shown the presence of phosphoric, dialkyl thiophosphoric, dealkyl thiophosphoric and dealkyl phosphoric acid (342). When applied to rice plants, methyl parathion gave rise to methyl paraoxon and to desmethyl methyl parathion (1021, 1022).

Paraoxon was detected, in samples of field-sprayed lettuce, four hours after application of parathion. Two other carboxyesterase inhibiting compounds, believed to be the S-ethyl and S-phenyl isomers of parathion, were also observed (290). In parathion-treated bean plants (Phaseolus vulgaris), paraoxon, p-nitrophenol, and possibly S-ethyl parathion were observed (402).

When roots of wheat plants were treated with parathion solutions, the leaf guttation fluid contained an active anticholinesterase which was shown to be paraoxon (342).

In the course of attempts to control Culex pipiens s.l., it was found that Bacillus subtilis was very effective in inactivation of parathion and methylparathion. A metabolite was identified as the amino analog of the original material (1529). Under culture conditions, B. subtilis metabolized methyl- and ethyl-parathion to their respective amino analogs. In additional studies with methylparathion, both desmethyl parathion and desmethyl-aminomethylparathion were observed. No oxon analog was detected (1018). In other studies, parathion was hydrolyzed by microorganisms to p-nitrophenol (626).

Soil bacteria decomposed parathion but metabolites were not isolated (1074). In soils of low numbers of microorganism (autoclaved soil) or of low microorganism activity (dry soils), parathion persisted for a relatively long time. No aminoparathion was found in autoclaved soils. Yeast reduced parathion in soil to aminoparathion (871).

Exposure of parathion to light resulted in the formation of cholinesterase inhibitors chromatographically different from parathion. The material resulting from the ultraviolet light treatment was a mixture of parathion, paraoxon, S-ethyl parathion, S-phenyl parathion and other oxidation and degradation products. That the mixture was less subject to diazotization and coupling suggested some destruction of the p-nitrophenol moiety. Exposure of the three identified products from parathion gave rise to many compounds, which did not separate into distinct spots on the chromatogram. Methyl parathion gave rise only to the corresponding paraoxon, which in turn gave rise to a less polar compound (302, 482, 626, 814, 815, 1139, 1267). Exposure of parathion to sunlight gave rise to p-nitrophenol (626). Parathion also underwent isomerization at elevated temperatures (406).

Hydrolytic studies indicated there was no catalysis by $[H^+]$. For parathion: $k = 0.047\ [OH^-] + 4 \times 10^{-6}\ min^{-1}$ (25°C.)

and $k = 0.14\ [OH^-] + 1.5 \times 10^{-5}\ min^{-1}$ (35°C.)

in 0.001N to 0.50N - NaOH

$t_{50\%}$ = 120 days in the absence of alkali (pH <10) (1141).

Para-nitrophenol, a degradation and hydrolytic product of parathion, was degraded by C. simplex. In cultures of pseudomonas with p-nitrophenol, hydroquinone and a quinone were reported to be present (575).

Degradation rates of dusts at various temperatures were fitted to Jander's rate equation, when the rate of degradation did not exceed 80% (1389).

$$kt = [1-(1-\alpha)^{1/3}]^2$$

α = reaction rate

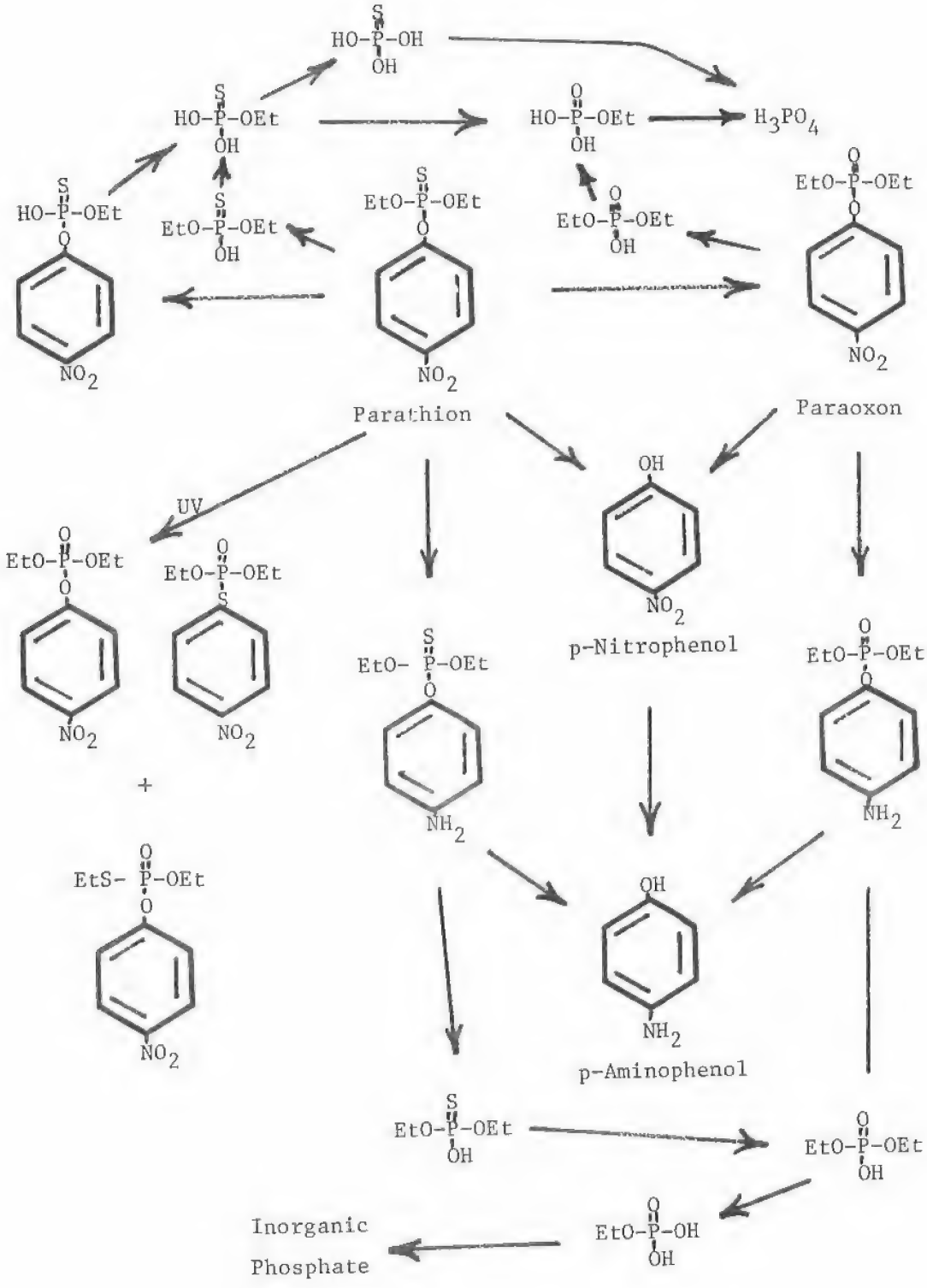
k = degradation rate constant

t = reaction time

Table III

T (°C)	k X 10^5
	Methyl Parathion dust
(Room temp.)	0.3
30	2.8
40	7.4
50	26
60	116

Under aseptic conditions, bean plants (Phaseolus vulgaris) apparently did not degrade parathion. When pure algal cultures were exposed to parathion or were added to the root chambers of the bean plants, amino analog was observed. Some p-nitrophenol was also detected (1781).


Parathion
Paraoxon
UV
p-Nitrophenol
p-Aminophenol
Inorganic
Phosphate

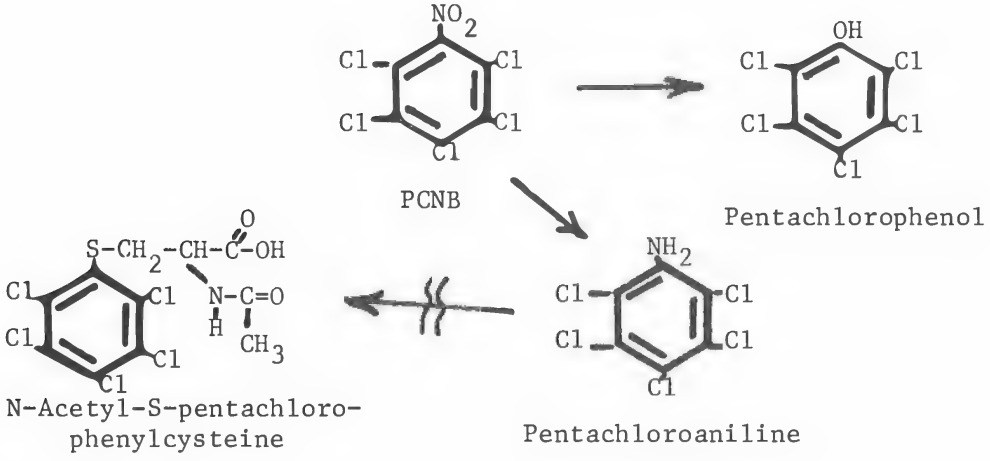
Pentachloronitrobenzene (PCNB)

PCNB was poorly absorbed in the gut of rabbits and underwent little reduction to the corresponding aniline until after absorption. The aniline analog was recovered from urine. N-acetyl-S-pentachlorophenylcysteine was also found. There was also evidence that some pentachlorophenol formed directly from PCNB (111).

Potatoes were grown in fields treated with PCNB. Analyses indicated that residues were mainly in the peel and that one metabolite was pentachloroaniline. A second metabolite obtained from the potatoes and a third from the soil were not identified. Evidence indicated that the metabolites were formed before penetrating the potato's peel (559).

Studies with fungi and microorganisms showed a rather widespread ability to convert PCNB to pentachloroaniline (258).

Fungi	Antinomycetes
Aspergillus niger	Norcardi sp.
Fusarium solani f. phaseoli	S. antibioticus
Glomerella cingulata	S. aureofaciens
Helminthosporium victoriae	S. cinnamoneus
Mucor ramannianus	S. griseus
Myrothecium verrucaria	S. lavendulae
Penicillum frequentans	S. venezuelae
Trichoderma viride	S. viridochromogenes



An isolate of Cephaloascus fragrans adapted to concentrations of PCP. Some degradation of PCP was observed with T. virgatum. No metabolites were identified (318).

Other studies indicated that disappearance of PCP in soils was more rapid in watered than in unwatered soils and that decomposition proceeded more rapidly in soils of high organic matter than in those of low organic content. Leaching did not appear to be important in the disappearance of PCP (1530).

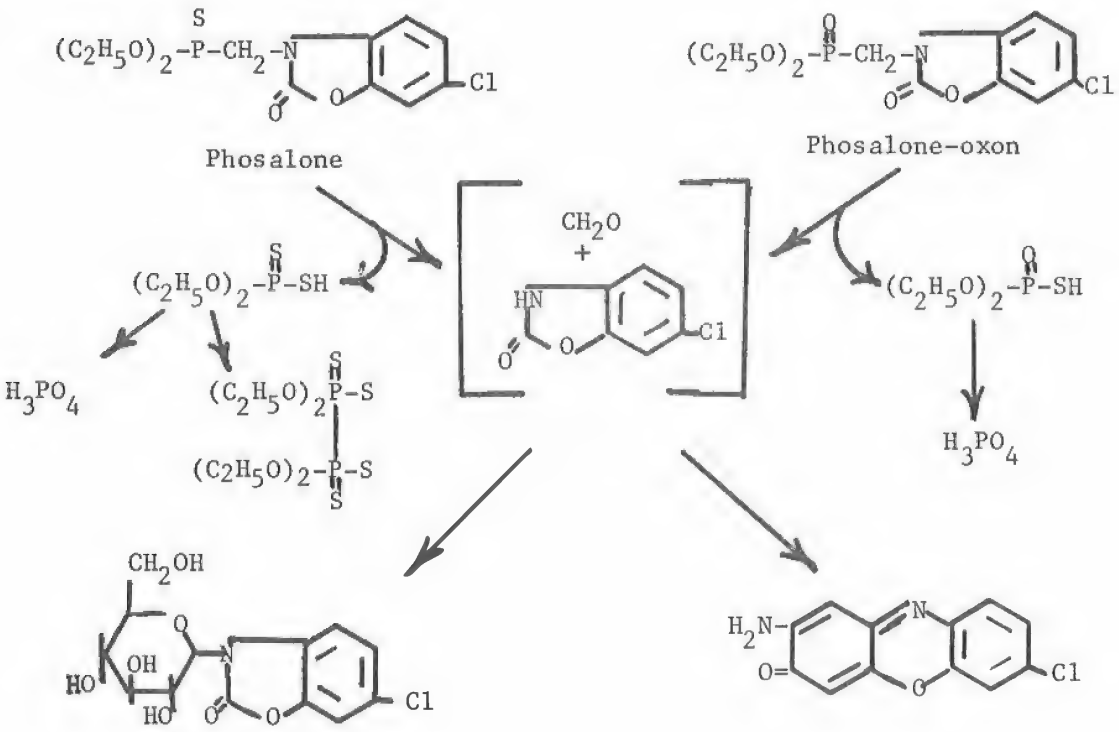
Studies showed that PCP was bound to muscle protein. Binding was markedly increased by decreasing pH from 9 to 7 and by addition of KCl, $MgCl_2$, and $(CH_4)_4$ NCl. Acetylation of actomysin greatly decreased the amount of bound PCP. This suggested that binding occurred on amino groups (146).

Phosalone [0,0-Diethyl S-(6-Chloro-2-oxo-benzoxazol-3-ylmethyl)phosphorodithioate]

Phosalone-oxon, which was formed in treated plants, degraded more rapidly than phosalone. In plants they were attacked hydrolytically at the P-S-C link. This gave the corresponding diethyl phosphorodithioic and diethylphosphorothioic acids and a benzoxazolone moiety. The latter was conjugated to form a glycoside (1792).

In soil, initial stages were apparently the same as in plants. However, a red material was observed and identified as substituted phenoxazone. A disulfide formed by oxidation of the phosphorodithioate (1792).

In rats, phosalone was rapidly metabolized when administered orally. Three unidentified compounds were present in the urine. When the carbonyl-C was labeled, 65.4% of the label appeared as $^{14}CO_2$ and 32.4% in urine and feces within four days (1792).

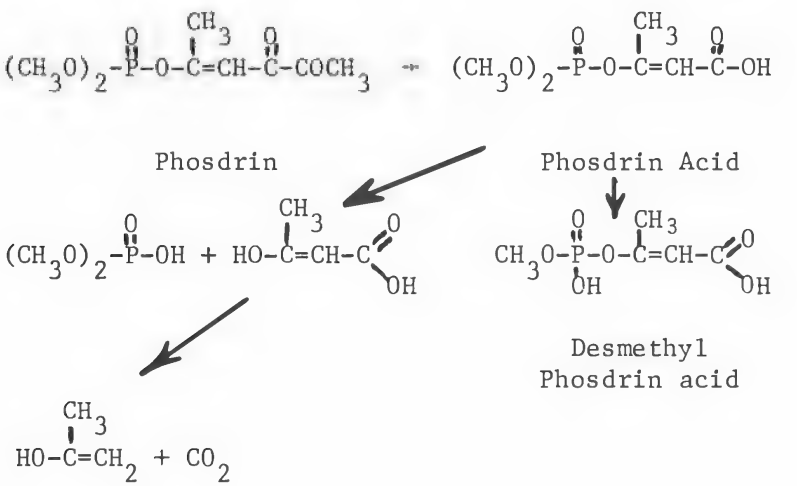


Phosdrin (0,0-Dimethyl 1-carbomethoxy-1-propen-2-yl phosphate)

When labeled phosdrin was fed to cows, blood levels of phosdrin metabolites reached a peak between 2 and 4 hours after administration and the blood was essentially free of radiation within 24 hours. Within three days, 77% of the administered dose was excreted. Half this was excreted via the urine within 12 hours. All radioactivity in the feces was present as hydrolysis products. Dimethyl phosphoric acid was identified in urine from a cow treated with phosdrin and in human plasma incubated with phosdrin (246).

In the presence of mouse liver enzymes and glutathion, cis-phosdrin was degraded to cis-desmethyl phosdrin and S-methylglutathione (1702). When the trans-isomer was tested, dimethylphosphate was obtained (1037).

Information on the metabolism of phosdrin in plants is not complete. However, the limited information available indicated that this compound was decomposed in plants by an initial enzymatic hydrolysis of the carboxylic ester to form the corresponding free acid. This was quickly followed by cleavage of the vinyl phosphate bond and decarboxylation. The finding of desmethyl phosdrin acid in some studies indicated that there were at least two paths for degradation of phosdrin in plants (245, 1341).



Phosphamidon [N,N-diethyl 1-Chloro-2-dimethylphosphate-prop-2-enamide]

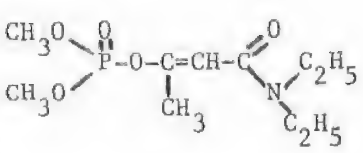
The comparative fate of the cis and trans isomers of phosphamidon was studied. The biological half-lives of the two isomers were very short in plants and animals and of comparable duration. Both isomers were converted to similar oxidative and hydrolytic metabolites. The rates of formation were different. Overall decomposition proceeded at almost equivalent rates (202).

After intraperitoneal injection of cis or trans phosphamidon-P^{32} in male white rats, excreta was collected during a 4-day period. About 60% of the dose--either isomer--was recovered within 24 hours after injection, most from urine. The hydrolytic products were almost entirely dimethyl phosphate, with small amounts of phosphoric acid and 0-demethyl phosphamidon (202).

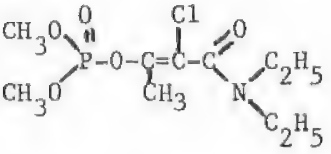
Phosphamidon was orally administered to white rats and a femal goat. In addition to hydrolysis products, nine metabolites were isolated from urine but only four were characterized: desethyl phosphamidon, deschlorophosphamidon amide, α-chloro-N,N-diethylacetoacetamide and an unsubstituted amide. Of these, only the desethyl metabolite was not found in milk (283).

Studies with labeled phosphamidon showed that plants metabolized phosphamidon to desethylphosphamidon, dimethyl phosphate, α-chloroacetoacetate diethyl amide and α-chloroacetoacetate ethylamide (29, 720, 985). After foliar treatment of cotton, phosphamidon, 0-demethyl phosphamidon, N-desethyl phosphamidon, hydroxyethyl phosphamidon, and dimethyl phosphate were detected externally. Internally, orthophosphate and an unknown were observed in addition to the foregoing compounds (202).

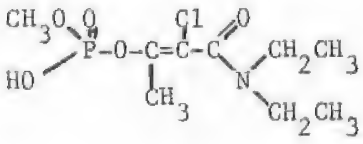
In fifth-instar tobacco budworms and bollworms, metabolism of the two isomers was similar. After injection of labeled phosphamidon, compounds I, II, III, IV, X, phosphamidon acid (VII), dimethyl phosphate (VIII), methyl phosphate (IX), and an unknown were observed in internal extracts. In the excreta, compounds I, II, III, IV, VII, VIII, and X were detected (202).



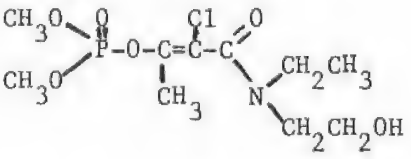
Deschlorophosphamidon amide (XI)



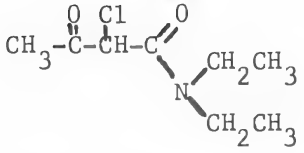
Phosphamidon (I)



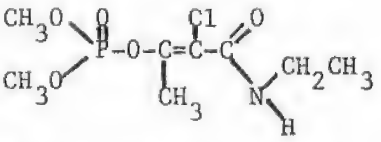
O-Demethyl phosphamidon (II)



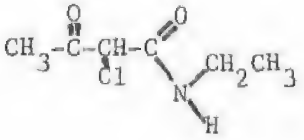
Hydroxyethyl phosphamidon (III)



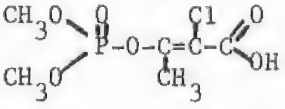
α-Chloro-N,N-diethylaceto-acetamide (V)



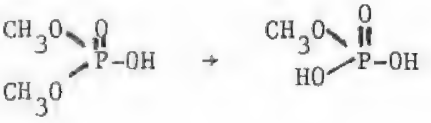
N-Desethyl phosphamidon (IV)



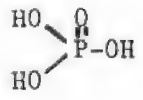
α-Chloro-N-ethylaceto-acetamide (VI)



Phosphamidon Acid (VII)



(VIII) (IX)



(X)

Picloram (Tordon) [4-Amino-3,5,6-trichloropicolinic acid]

Carboxy-labeled picloram was applied to cotton plants and the metabolism studied. Only labeled CO_2 was identified as a metabolite. Hydrolysis with 6 N-HCl of unextractable radioactivity indicated that about 3% of the tordon was probably combined with terminal amino groups of protein in the cotton plant (971, 972).

Wheat seedlings were grown in soil treated with picloram. Analysis of plant material indicated the presence of oxalic acid, 4-amino-3,5-dichloro-6-hydroxypicolinic acid, unchanged picloram and lipids hydrolyzable to all three acids and 4-amino-2,3,5-trichloropyridine (1199, 1201).

Attempts to isolate organisms that could utilize picloram as a good energy source were unsuccessful. However, a number of organisms were found capable of degrading this herbicide if a supplementary energy source were made available (1533).

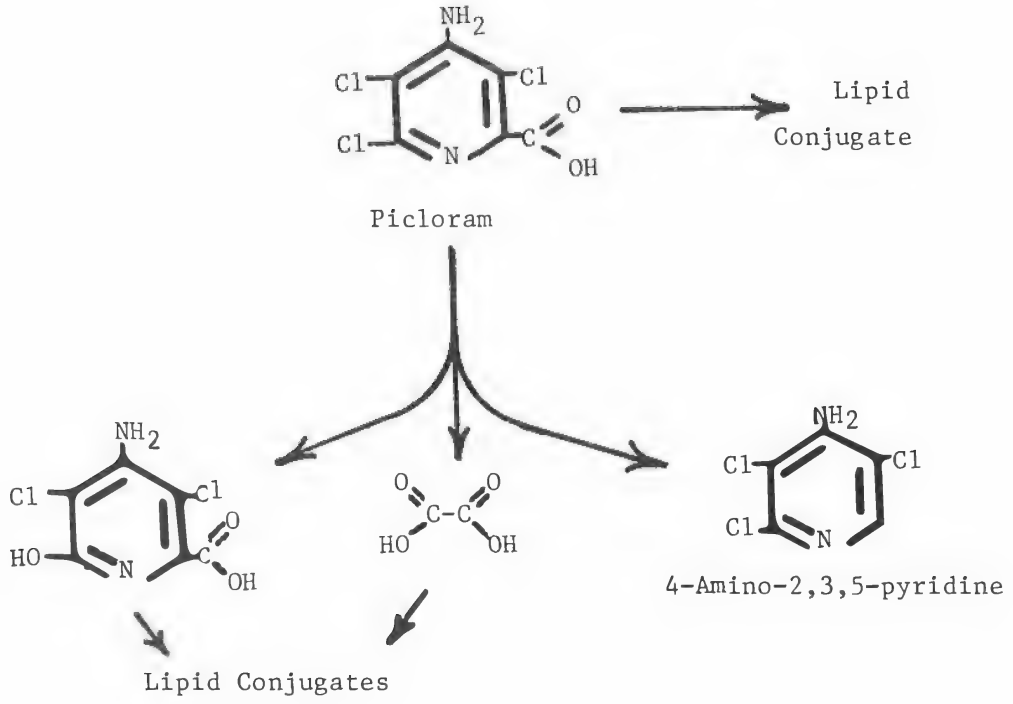
Bacillus cereus
Bacillus subtilis
Nocardi opaca (ATCC-4276)
Agrobacterium radiobacter (ATCC-4718)
Arthrobacter globiformis (ATCC-8010)
Flavobacterium aquatile (ATCC-11947)
Aspergillus niger (ATCC-6275)
Aspergillus terreus
Botrytis alii
Penicillium digitatum
Trichoderma lignorum
Saccharomyces cerevisiae
Helminthosporium pedicellatum

Degradation studies of picloram in soil were conducted.

$$t_{1/2} = 0.693/k$$

In Regina heavy clay, at 25°C and at moisture holding capacity, $t_{1/2}$ = 68 ± 10 days (569).

Picloram was degraded rapidly by short wave ultraviolet light but slowly by sunlight (986).



Prolan [Bis(p-chlorophenyl)-2-nitropropane]

Sublethal doses of prolan were metabolized to unidentified compounds. In addition to unchanged prolan, an acidic metabolite, possibly bis(p-chlorophenyl)pyruvic acid, has been isolated from the excreta of houseflies. UV and I.R. absorption and chemical tests showed that the NO_2 group had been lost and that a >C=O was present. It has been shown that this is not DDA (1142, 1145).

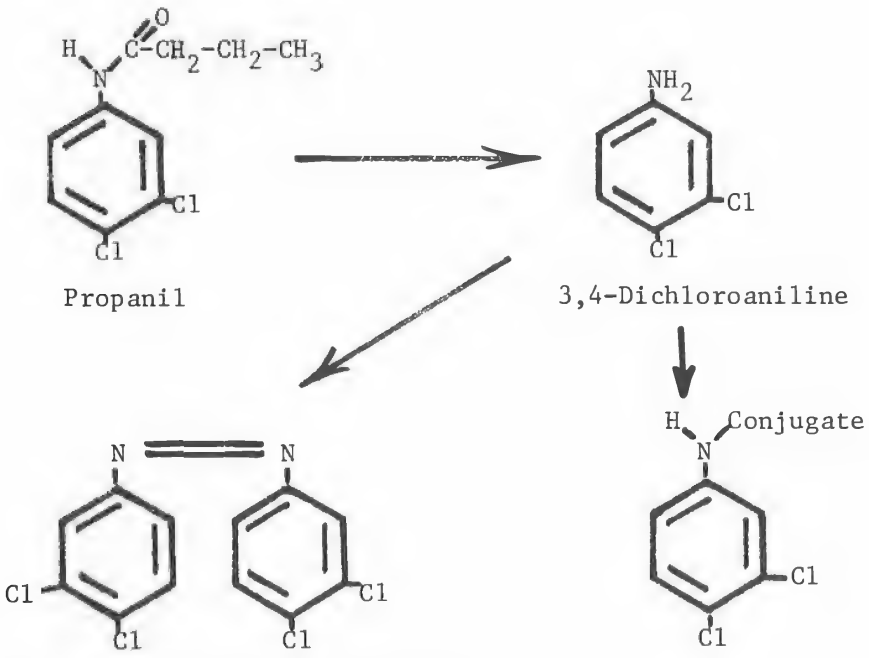
Propanil (DCPA, Stam-F 34, DPA) [N-(3,4-Dichlorophenyl)propionamide]

Liver homogenates from rats, mice, rabbits, and dogs contained an enzyme which hydrolyzed propanil to form 3,4-dichloroaniline (1502).

In intact rice plants, propanil was metabolized to four compounds. Two were identified as 3,4-dichloroaniline and N-(3,4-dichlorophenyl) glucosylamine. The latter was apparently converted to other unidentified metabolites (1363). In other studies, in addition to the glucosylamine, dichloroaniline was found combined as a saccharide containing glucose, xylose and fructose and combined with lignin (1618). Plants metabolized the propionic acid moiety to CO_2 (1597).

From cell free extracts of plants, an acylamidase system was obtained that transformed propanil into 3,4-dichloroaniline and five other metabolites (1364, 1365).

In soils, propanil was degraded to dichloroaniline and 3,3'4,4'-tetrachloroazobenzene (89, 1627, 1649). A fungus was isolated that could utilize propanil as the sole source of organic carbon. CO_2 was formed by oxidation of the propionate moiety. Dichloroaniline was also a product of the transformation but did not persist (1674).

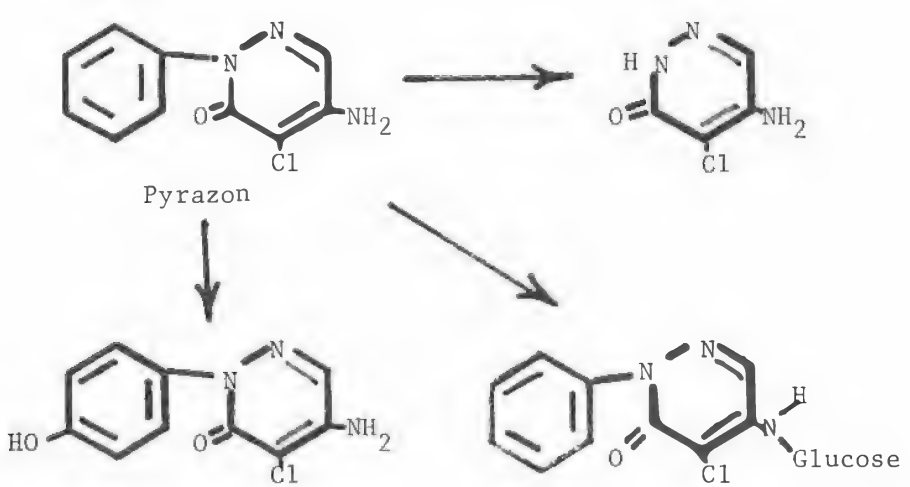


Pyrazon (Pyramin, Pyrazonl, PCA, HS-119) [5-Amino-4-chloro-3-oxo-2-phenyl-(2H)-pyridazine]

The urine of rabbits fed with pyrazon was hydrolyzed and analyzed by means of thin-layer chromatography. Approximately half the amount of pyrazon administered orally to rabbits was excreted in the urine within 24 hours of ingestion in the form of the unchanged chemical and its metabolite the 2-(p-hydroxyphenyl) analog. Urinary excretion products resulting from oral administration of tech. pyrazon included not only unchanged pyrazon and the p-hydroxy metabolite, but also isopyrazon, present in the tech. formulation as an impurity, and its p-hydroxy metabolite. Similar results were obtained with cats and dogs, although in contrast to rabbits more p-hydroxy metabolite was excreted than unchanged pyrazon (973).

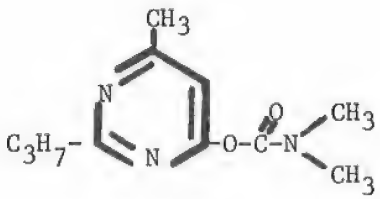
Studies with pyrazon indicated that 5-amino-4-chloro-3-oxo-(2H)-pyridazine was formed after application to beets (456). A second metabolite was also observed in subsequent studies but was not identified (1353). Resistant red beets, Beta vulgaris L., were treated with pyrazon tritiated in the phenyl ring. A combination of gas liquid chromatography, infrared spectroscopy and thin layer were used to identify the metabolite as the N-glucosamine of pyrazon (1211).

4-Amino-5-chloro-3-oxo-(2H)-pyridazine was also found in soil treated with pyrazon (456).

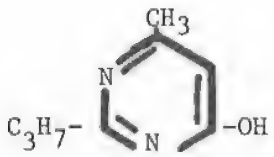


Pyromat [4-methyl-2-propyl-6-oxypyrimidyl-N,N-dimethylcarbamate]

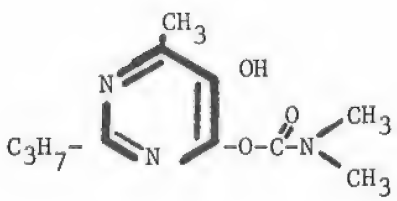
After extraction of urine, paper chromatography and chemical reactions were used to determine those metabolites that were present. Metabolite I was identified as 4-methyl-2-propyl-6-hydroxypyrimidine (m.p. 144°C) after preparation from pyromat by hydrolysis. Studies with mice, rats, guinea pigs, and rabbits indicated that metabolite III arose from the loss of the carbamate group from metabolite II. Metabolite III was believed to be 4-methyl-2-propyl-5,6-dihydroxypyrimidine. After oral feeding of II to rats, only III and unchanged II were detected, thereby demonstrating that two paths of degradation existed (1189).



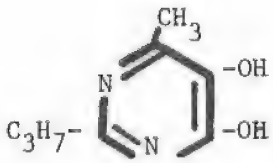
Pyromat



I



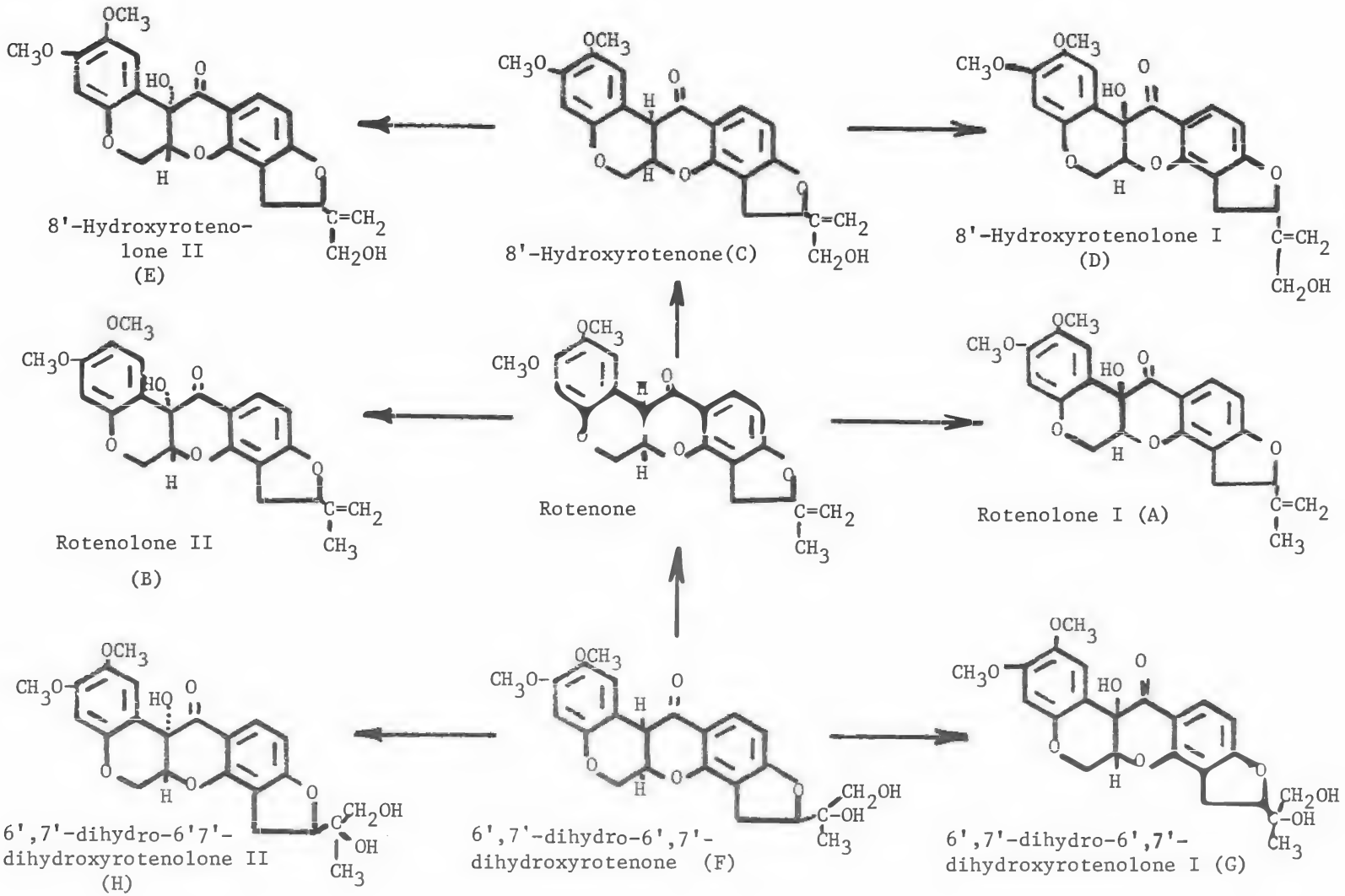
II



III

Rotenone

Microsome fractions from housefly abdomens, mouse livers, and rat livers were used to study rotenone degradation. Metabolites so produced were in part the same as in living mice and flies. I.R., U.V., chromatography, chemical reactions, and mass spectra were used to identify metabolites. Sequence studies demonstrated the relation of the metabolites: A, B, C and F arose from rotenone but not from any other intermediate. A yielded D and G; B yielded E and H; C yielded D and E; F yielded G and H (502, 1527).

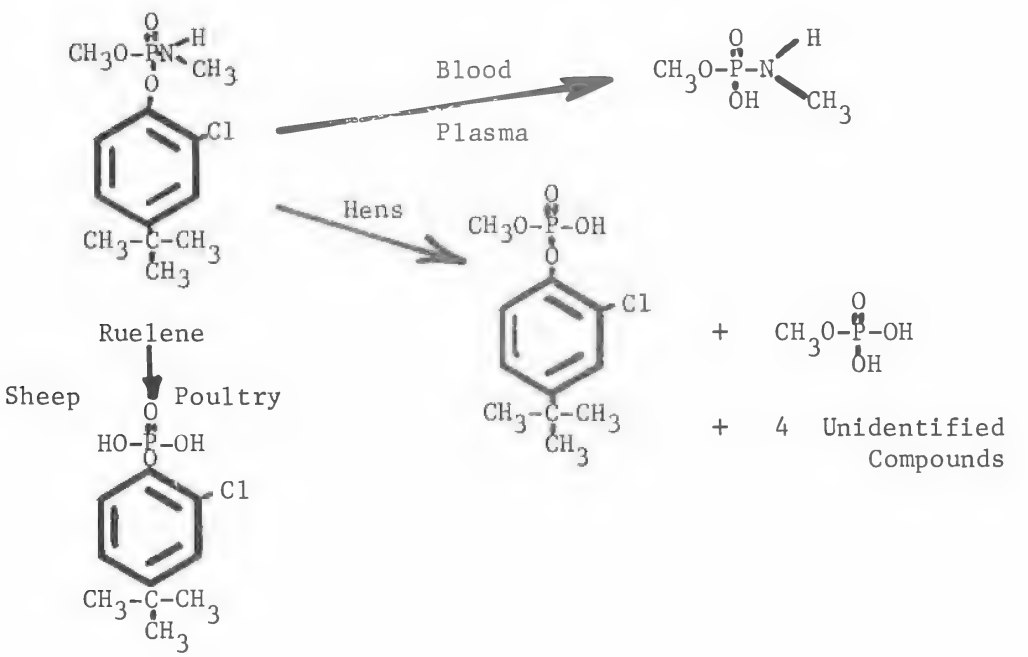

8'-Hydroxyroteno-
lone II
(E)
8'-Hydroxyrotenone(C)
8'-Hydroxyrotenolone I
(D)
Rotenolone II
(B)
Rotenone
Rotenolone I (A)
6',7'-dihydro-6'7'-
dihydroxyrotenolone II
(H)
6',7'-dihydro-6',7'-
dihydroxyrotenone (F)
6',7'-dihydro-6',7'-
dihydroxyrotenolone I (G)

Ruelene [4-tert-butyl-2-chlorophenyl methyl methylphosphoroamidate]

Excretion and blood data, obtained after sheep were given oral doses of ruelene-P^{32}, indicated that ruelene is rapidly absorbed and excreted. Eighty-five to ninety-five per cent of the dose was recovered in the excreta. Analyses showed that this compound was degraded to inorganic phosphate which was subsequently incorporated in bone. After incubation of ruelene with whole blood and plasma, chromatography showed the presence of dimethylamido phosphoric acid and phenylmethyl phosphoric acid. Several other metabolites were found but not completely identified (94).

Sheep and poultry hydrolyzed both ester and amide groupings and formed a large number of innocuous hydrolytic compounds that were excreted via urine of sheep or urine and feces of poultry. One compound from sheep urine was believed to be 4-t-butyl-2-chlorophenyl dihydrogen phosphoric acid. A compound was isolated from insects that had a similar R_f (66).

Ruelene was fed to laying hens at a level of 100 p.p.m. for seven days. Six metabolites were isolated from excreta. Of these O-methyl-phosphoric acid and 4-tert-butyl-2-chlorophenyl methyl phosphoric acid were tentatively identified. Residues appeared in egg yolks 3 - 5 days after return to normal feed. Some tissues were residue-free after three days (213).



Schradan (OMPA, octamethylpyrophosphoramide) [bis-[dimethylamino]-phosphonous anhydride]

Using the rate constant for the hydrolysis of schradan (at 25°C., as $4.8 \times 10^{-3} \times [H^+]$ min^{-1}), calculations indicated that 312 and 1040 days would be required for 50% and 90% hydrolysis, respectively, in Valencia oranges, and 16 and 35 days, respectively, in lemons. Values obtained after actual treatments indicated a gradual breakdown of schradan but not as rapid as theory suggests. Evaporation loss has been calculated to be 1.5×10^{-3} $mg/hr/cm^3$ at 15°C (996).

Metabolic degradation of schradan in plants, animals, and insects gave rise to the same highly active and anticholinesterase compound (34, 109, 272, 343, 350, 351, 381, 382, 524, 525, 550, 551, 619, 621, 622, 623, 637, 638, 1090, 1091, 1097, 1098, 1346, 1402, 1403). Using countercurrent distribution data, solvent partitioning characteristics enzymatic inhibitor assays, and chemical reactions, the data indicated that this metabolite is the monophosphoramide oxide of schradan (239, 240, 241, 243). This underwent isomerization with subsequent loss of formaldehyde and formation of heptamethylpyrophosphoramide. These studies indicated that, while formation of the N-oxide is the primary pathway, some hydrolysis of the phosphate also occurred (639).

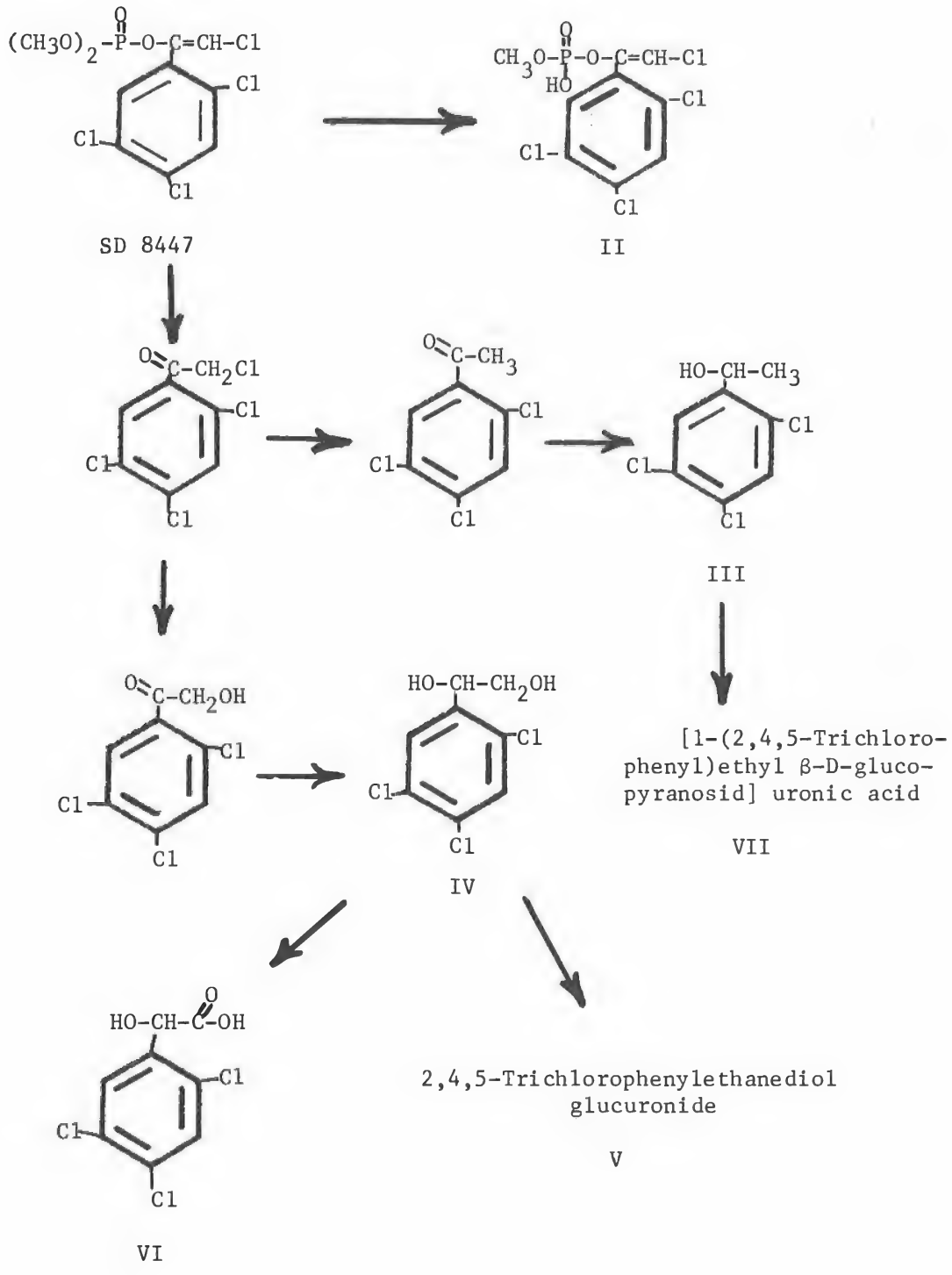
In the locust (Schistocerca gregaria Forsk.) fat body, the production of an esterase inhibitor was dependent upon the presence of particulate and soluble fractions of the cell contents, required oxygen, and was activated by Ca^{++} or Mg^{++} ions (445).

SD 8447 [2-Chloro-1-(2,4,5-trichlorophenyl)vinyl dimethyl phosphate]

P^{32}-labeled SD 8447 in soybean oil was administered via stomach tube to male and female Wistar rats. There was rapid metabolism to hydrolysis products of low toxicity to mammals. P^{32} in the urine was present as dimethyl hydrogen phosphate, methyl dihydrogen phosphate and desmethyl SD 8447 (1493).

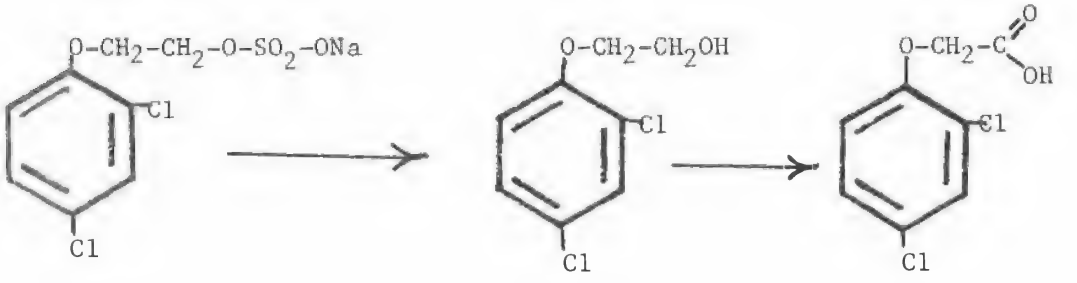
Single doses were almost completely eliminated by rats in four days with 78% of the label appearing in the urine, 16.5% in feces, and 0.5% in expired gases. In dogs, after oral administration, 92% of the label appeared in the urine during four days. Metabolic products identified in the urine of rats and dogs were, respectively (11): 2,4,5-trichlorophenylethanediol glucuronide (V) (8, 12%); [1-(2,4,5-trichlorophenyl)ethyl β-D-glucopyranosid] uronic acid (VII) (35, 0%); 2,4,5-trichloromandelic acid (VI) (24, 12%); 2-chloro-1-(2,4,5-trichlorophenyl)vinyl methyl hydrogen phosphate (II) (4, 46%); 2,4,5-trichloroethanediol (IV) (2.5, 4%); and 1-(2,4,5-trichlorophenyl)ethanol (III) (2, 0%).

Recent studies have shown that this compound reacts with and is bound by proteins in the blood plasma of mammals and in homogenates of housefly heads (155).


SD 8447
II
III
[1-(2,4,5-Trichloro-
phenyl)ethyl β-D-gluco-
pyranosid] uronic acid
VII
IV
2,4,5-Trichlorophenylethanediol
glucuronide
V
VI

Sesone (2,4-Dichlorophenoxyethylsulfate)

At a pH of 3.0 - 4.0, the ethanol analog of 2,4-dichlorophenoxyethylsulfate was formed by hydrolysis. Above pH 5.5, this reaction ceased unless microflora were present. In the presence of Bacillus Cereus var. Mycoides, activation proceeded via enzymatic hydrolysis and oxidation probably to 2,4-dichlorophenoxyacetate (47, 49, 228, 229, 1452, 1453, 1454).



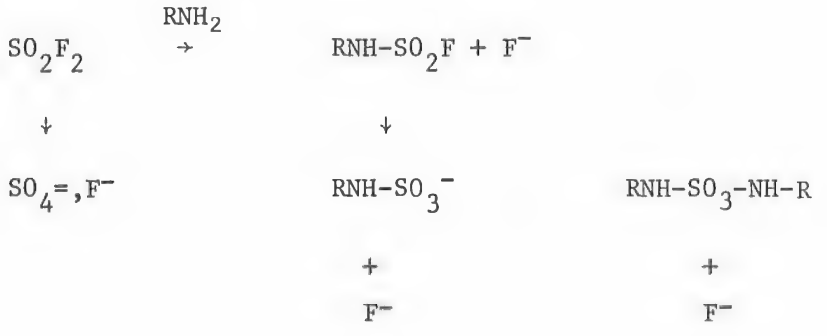
Sesone | Sesone Alcohol | 2,4-D

Sulfur

The basis of toxic action of sulfur is not clear. It is reduced to H_2S by fungal spores but this does not account for its toxicity (1009). It has been suggested that sulfur acts as a hydrogen acceptor in metabolic systems, thereby disrupting normal hydrogenation and dehydrogenation reactions. It has also been suggested that polysulfide free radicals may form cross linkages with protein and other cellular components. Studies have also shown that sulfur blocks enzymes in the pathway between acetate and citrate and partially in the succinoxidase systems (1120, 1310).

Sulfuryl Fluoride (SO_2F_2)

Under conditions of usage as a grain fumigant, sulfuryl fluoride reacted with protein and released inorganic fluoride. Some sulfate was also found (968)



Sumiphonothion [O-methyl O-3-methyl-4-nitrophenyl methylphosphonothioate]

P^{32} labeled sumiphonothion was fed to Swiss mice. Examination by ion-exchange chromatography of collected urine and feces revealed seven compounds: sumiphonothion oxon and its desmethyl analog, desmethyl sumiphonothion, O-methyl methylphosphonothioate, O-methyl methylphosphonate and methyl phosphonate. One other compound was found but not identified (682).

Sumithion (Fenitrothion) [0,0-Dimethyl O-(3-methyl-4-nitrophenyl) phosphorothioate]

Activation studies of sumithion were conducted with slices and tissue homogenates of mammals, insects, and plants. Ion exchange chromatography indicated that although the oxon analog is formed, desmethyl sumithion is the principal metabolite. Some phosphoric or thiophosphoric dialkyl thiophosphoric and dialkyl phosphoric acids were also liberated (1016, 1017, 1021, 1022, 1443, 1687).

After exposure of rats and guinea pigs to sumithion, the desmethyl analog, dimethyl phosphorothioate, dimethyl phosphate, and four unidentified compounds were found (1021, 1022).

Urine and feces, from male Swiss mice fed P^{32} labeled sumithion, was collected and chromatographed on paper. Dimethyl phosphate was the major metabolite found at all dosage levels. In addition to this, six compounds were observed and identified as orthophosphate, methyl phosphate, sumioxon, dimethyl phosphorothioate, desmethyl sumioxon and desmethyl sumithion. An eighth compound was observed but not identified (682).

A 3% dust of sumithion, at the rate of 100 mg/kg/day for 50 - 90 days was administered orally to cattle, sheep, and pigs. No sumithion residues were detected in the milk but large amounts of sumithion, sumioxon, and p-nitrocresol were found in the urine of milk cows, steers, and sheep. When a 25% emulsion of sumithion was administered orally at the rate of 10 mg actual sumithion/kg/day for five days to milk cows, small amounts of sumithion and sumioxon were found in the milk (1071). In other studies with Jersey cows, aminosumithion was also found in the milk (1583). (This could have been formed by rumen bacteria.)

P^{32} labeled sumithion was applied topically on the pronotum of 2-day old female flies, both resistant and non-resistant. Resistant flies detoxified sumithion at a more rapid rate than susceptible flies. However, there was no qualitative difference in the metabolites found. In addition to one unidentified compound, orthophosphate, mono- and di-methyl phosphate, sumioxon, phosphorothioate, dimethyl phosphorothioate, desmethyl sumioxon, and desmethyl sumithion were found (683).

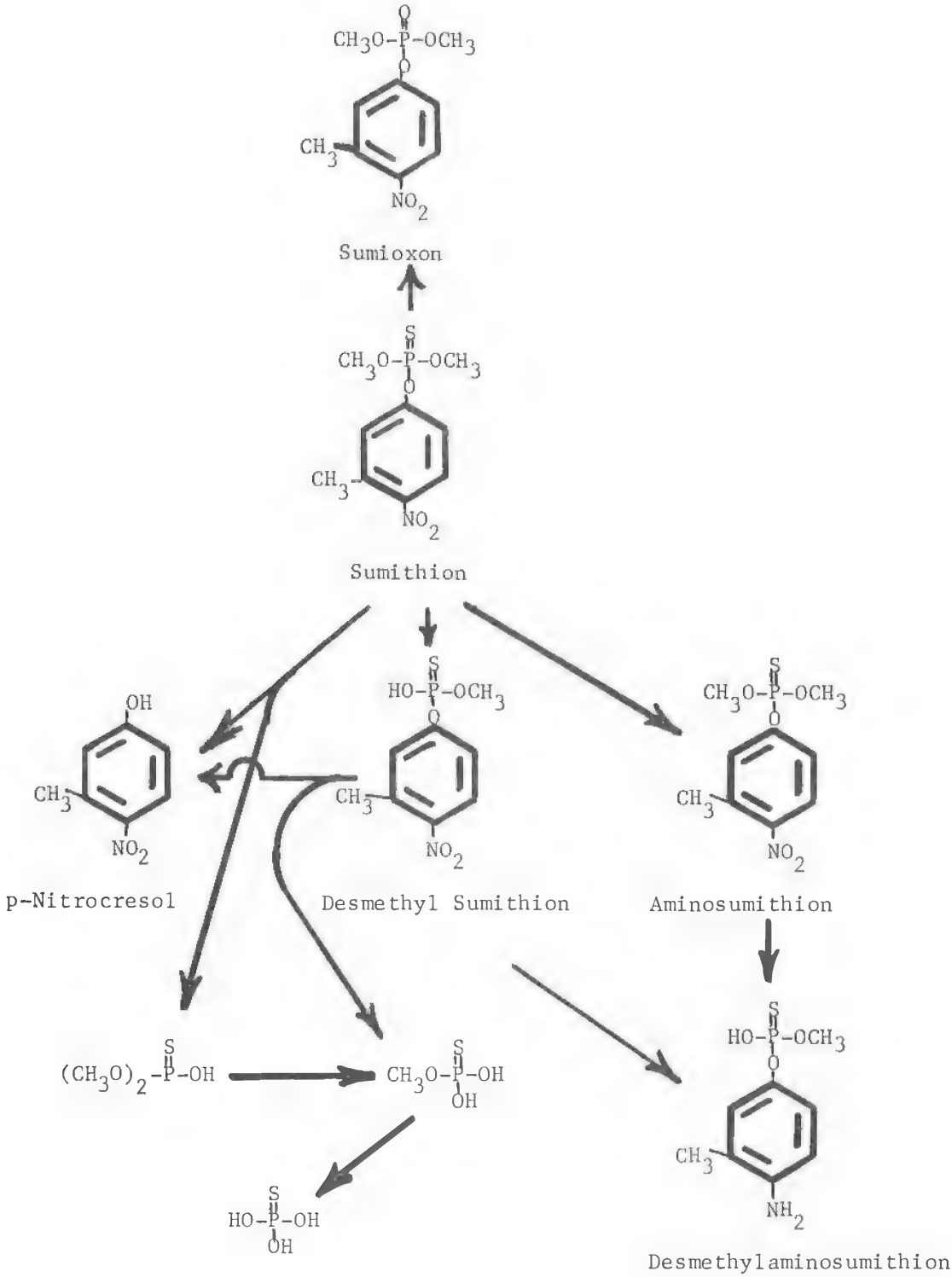
Studies with Bacillus subtilis, isolated from polluted water samples, showed that it was capable of inactivating sumithion by reduction of its nitro group. The metabolite was identified as amino-sumithion (1529).

Under culture conditions, Bacillus subtilis metabolized sumithion primarily to the amino compound. This was metabolized, at a lower rate than the parent compound, to desmethylaminosumithion. Desmethylsumithion, and dimethylphosphorothioic acid were also observed. Desmethylaminosumithion arose from aminosumithion. Dimethylphosphorothioate came from sumithion. No sumioxon was detected (1018).

When P^{32}-labeled sumithion was applied to rice plants at the preheading stage, the sumithion penetrated into the tissues and was rapidly degraded to desmethylsumithion, dimethyl phosphorothioic acid and phosphorothionic acid. Sumioxon was present and minute amounts of such degradation products as dimethyl phosphorothioic acid, phosphorothionic acid and free p-nitrocresol were identified. The latter compound was also found in cocoa beans exposed to sumithion (1019, 1020).

When German cockroaches were treated with sumithion, the desmethyl analog was found (1021, 1022).

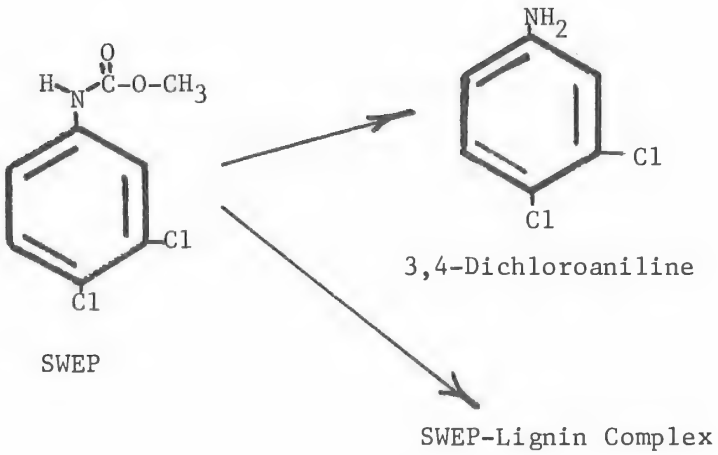
Decomposition of sumithion at 100 - 140°C produces a mixture containing mostly organophosphorus polymers. O,S-dimethyl O-3-methyl-4-nitrophenyl phosphorothioate was formed as an intermediate which decomposed further to 3-methyl-4-nitrophenol and polymers. Sumioxon was not observed (1578).


Sumioxon
Sumithion
p-Nitrocresol
Desmethyl Sumithion
Aminosumithion
Desmethylaminosumithion

SWEP [Methyl N-[3,4-dichlorophenyl] carbamate]

In green house studies SWEP was applied to rice, carrots, oats, wheat, corn, and barnyard grass. There were indications that trace amounts of SWEP were hydrolyzed in soil to 3,4-dichloraniline. However, it was found that most of the herbicide was immobilized and bound as a lignin complex (274).

(See also Karsil, Propanil, Dichloran, and Ramrod.)



TCA [Trichloroacetate]

C^{-14}-labeled trichloroacetate was applied to corn and pea plants. TCA itself, but no metabolic products, was found (123).

In soil, microorganisms degraded trichloroacetate after a lengthy period of adaptation. Dechlorination occurred with probable formation of oxalate (666, 731, 732).

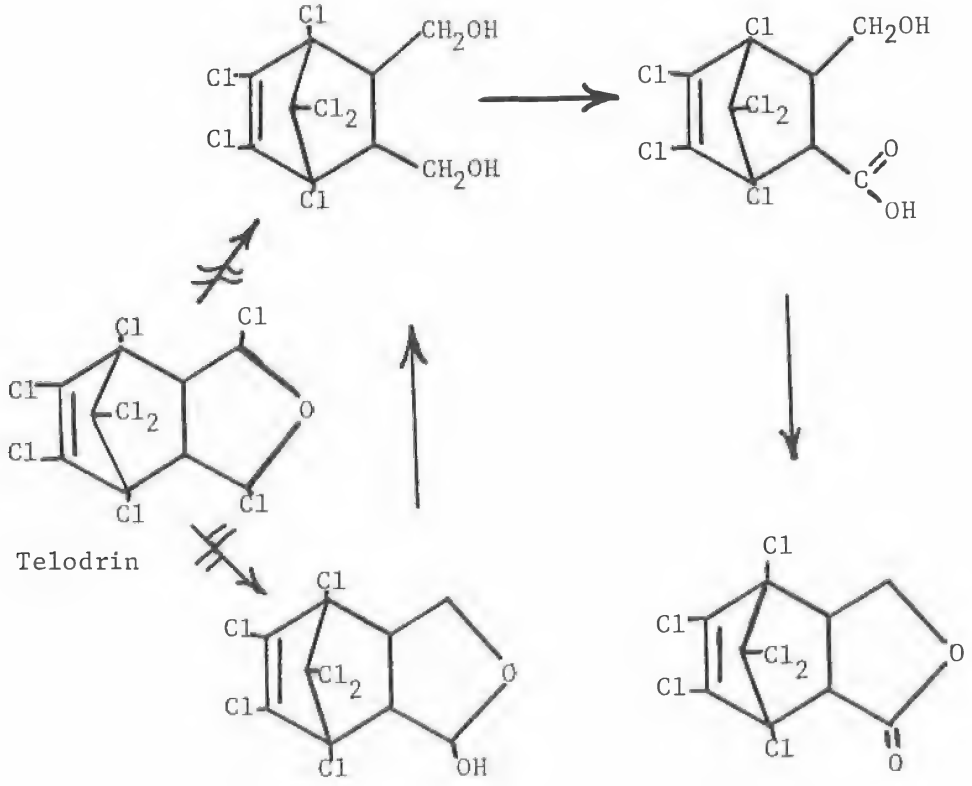
Breakdown of TCA by Pseudomonas dehalogenans and a Norcardia sp. was shown by evolution of chloride (665, 667, 668).

Telodrin [1,3,4,5,6,7,8,8-Octachloro-3a,4,7,7a-tetrahydro-4,7-methanophthalan]

The secretion of telodrin or its metabolites was studied in cows by means of G. L. C. - electron capture. Residues of telodrin were found in the milk and meat of the cows in proportion to the level fed (118).

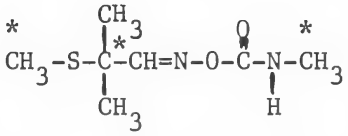
Mosquito larvae were exposed to C^{-14}-labeled telodrin and four metabolites were obtained. Using paper chromatography, it was found that chlorine in the 1 and 3 positions was split off and the compound was identified as a lactone (830, 831).

When the metabolism of telodrin by microorganisms was studied, it was found that the molds, Aspergillus niger and flavus and Pencillum chryssogenum and notatum, could degrade telodrin. A hydrophyllic metabolite was isolated but not identified. This appeared to be the same as one of the metabolites obtained when mosquito larvae were exposed to telodrin (831).



Temik [2-Methyl-2-(methylthio)propionaldehyde O-(methylcarbamoyl)oxime]

Male rats (Carworth Farms-Elias stock) were treated orally and intraperitoneally with labeled Temik in ethanol and Temik sulfoxide in water. The excretion of S-methyl-C^{14} (1) and tert-butyl-C^{14} (2) temik was completed, essentially, in four days. The N-methyl-C^{14} (3) was excreted in urine and feces up to 11 days. In collected urine, four metabolites were identified as: 2-methyl-2-(methylsulfinyl)propionaldehyde oxime (III), 2-methyl-2-(methylsulfinyl)propionaldehyde O-(methylcarbamoyl) oxime (V), 2-methyl-2-(methylthio) propionaldehyde oxime (II) and 2-methyl-2-(methylsulfonyl) propionaldehyde oxime (IV). Three other metabolites were not identified (810).



(1) (2) (3)

In other studies, 80% of the dose was rapidly excreted in the urine and 4% via feces within 24 hours. Half of the metabolites in the urine were not extractable into organic solvent and remained unidentified. The remainder was tentatively identified as:

2-methyl-2-methylthiopropionaldoxime (II);

2-methyl-2-methylsulfinylpropionaldoxime (III);

2-methyl-2-methylsulfonylpropionaldoxime (IV);

2-methyl-2-methylsulfinylpropionitrile (VIII);

2-methyl-2-methylsulfonylpropionitrile (IX);

2-methyl-2-(methylsulfinyl)propionaldehyde O-(methylcarbamoyl) oxime (V);

and 2-methyl-2-(methylsulfonyl)propionaldehyde O-(methylcarbamoyl) oxime (VI) (24, 26).

In vitro studies with subcellular fractions of rat livers were conducted. TLC was used to isolate metabolites. Compounds II, IV, V and VI were identified (24, 26, 1110, 1112).

The major route of elimination of the Temik-S^{35} dose administered to the lactating cow was via urine. Approximately 83% of the dose was eliminated within 24 hours and 90% within 540 hours. Total radioactivity in the milk accounted for 3.02% of the dose. Only 2.85% of the Temik -S^{35} was eliminated in the feces. Extracted and tentatively identified in the milk were eleven compounds, including temik sulfoxide (V) and sulfone (VI), oxime sulfoxide (III) and sulfone (IV), nitrile sulfoxide (VII) and sulfone (VIII), temik oxime (II), and four unidentified compounds. Metabolites identified in the urine were qualitatively identical but differed quantitatively. Only five metabolites were identified in the feces: compounds II, III, V, VI and the nitrile sulfone (VIII) (1564).

Rats were fed milk from cows treated with temik-S^{35}. Analyses indicated six compounds not identified in addition to temik sulfoxide (V) and sulfone (VI), oxime sulfoxide (III) and sulfone (IV), nitrile sulfoxide (VII) and sulfone (VIII), and temik oxime (II) (1564).

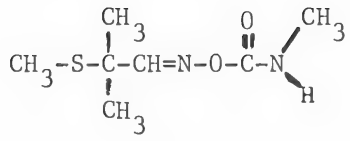
Female flies (Musca domestica S_{NAIDM} and R_{MIP} strains) were treated topically with labeled temik. From the internal C^{14} in the flies, the identity of temik, temik sulfone and temik sulfoxide was confirmed with TLC. Temik oxime arose through hydrolysis with concurrent formation of methylcarbamic acid. The latter gave rise to CO_2. The latter also arose from N-dealkylation of the N-methyl group or by oxidation of the S-methyl group after S-dealkylation. Another compound was thought to be the hydrolysis product of temik oxime sulfone. Examination of feces showed that the major excretory product was temik sulfone. No evidence of temik sulfoxide or temik sulfone was observed (24, 26, 1001).

S^{35}-Temik was applied topically to boll weevils (Anthonomus grandis Boheman). Externally, little hydrolysis occurred but oxidation to the sulfoxide was substantial. In excreta and internal extracts, twelve unidentified metabolites were observed in addition to Temik sulfoxide, the oxime sulfoxide, and the two corresponding sulfones (24, 26, 201).

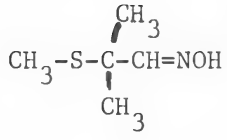
After injection of labeled temik into tobacco budworms (Heliothis virescens F.), a number of unidentified metabolites were observed in addition to temik sulfoxide and sulfone, the oxime sulfoxide and sulfone, and nitrile sulfoxide and sulfone. Trace amounts of products that cochromatographed with N-demethyl temik sulfoxide and sulfone were also detected (201).

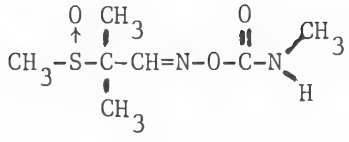
Large isolated cotton leaves were treated with labeled temik. Temik disappeared rapidly. Temik sulfoxide appeared as the major metabolite with a small amount metabolized to the sulfone. By way of hydrolysis, Temik oxime was the primary hydrolytic product. The hydrolysis product of Temik sulfone or Temik oxime sulfone was also present (991).

Under laboratory conditions, temik was degraded more slowly in soil than in cotton but similar metabolites were observed in both. In cotton, temik sulfoxide and sulfone, the nitrile sulfoxide and sulfone and oxime sulfoxide were observed. In soils studied, temik sulfoxide and sulfone, nitrile sulfoxide, oxime sulfoxide, and the oxime were observed. In both test systems, approximately five additional products were unidentified (305). Field studies confirmed the laboratory studies. In soil, however, much temik was lost by volatilization and/or leaching (1743).



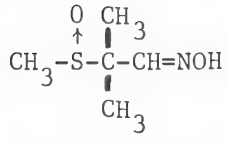
Temik (I)

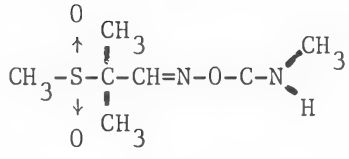




Temik oxime (II)

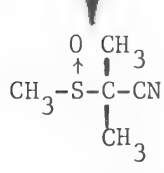
Temik sulfoxide (V)

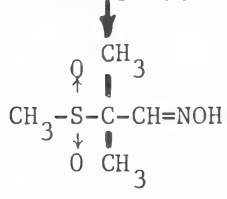




Oxime sulfoxide (III)

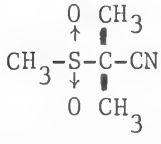
Temik sulfone (VI)





Nitrile sulfoxide (VII)

Oxime sulfone (IV)



Nitrile sulfone (VIII)

T.E.P.P. (Tetraethyl pyrophosphate)

T.E.P.P. is soluble in water. At concentrations normally employed, hydrolysis is of the first order provided pH is constant or on the acid side.

$$k = \frac{1}{t} \log_e \frac{a}{a-x} = \frac{2.303}{t} \log_{10} \frac{a}{a-x}$$

Since hydrolysis is not appreciably catalysed by hydrogen ions, at 25°C, k reduces to:

$$k = 160[OH^-] + 1.6 \times 10^{-3} \text{ min.}^{-1}$$

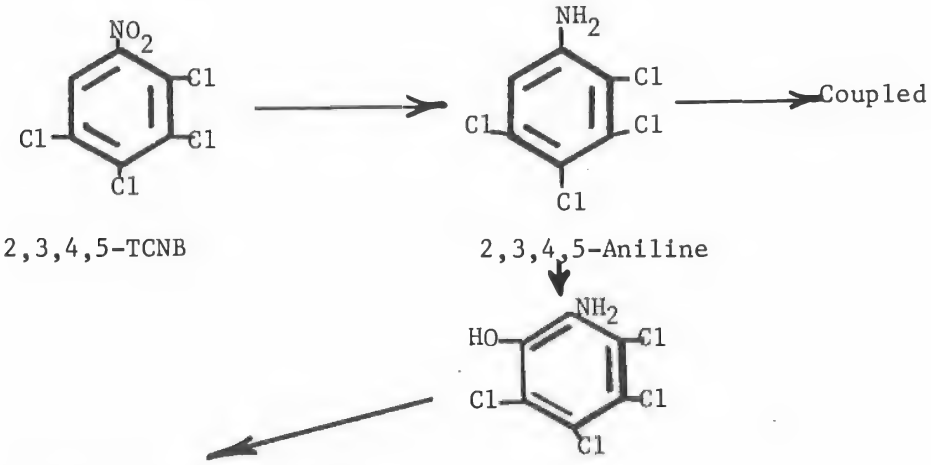
For convenience, the rate of hydrolysis in terms of the time for 50% decomposition is expressed:

$$t_{50\%} = \frac{\log_e 2}{k} = \frac{0.693}{k}$$

Tetrachloronitrobenzene (TCNB)

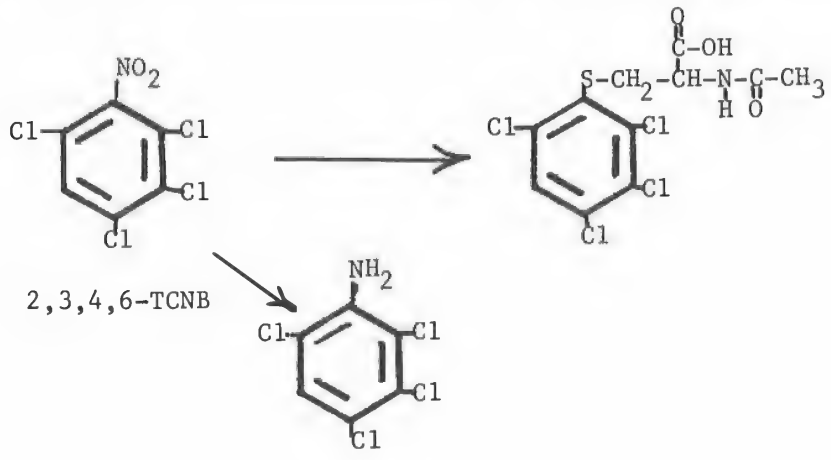
2,3,4,5-TCNB

This isomer exhibited greater absorption in rabbits than the 2,3,5,6-isomer. Unabsorbed 2,3,4,5-TCNB was reduced, in large measure, in the gut to the amine which diazotizes readily and couples. Absorbed TCNB was partly converted to a glucuronide (27% of dose) and to an ethereal sulfate (7% of dose). 2,3,4,5-aniline was isolated from urine. No mercapturic acid formation was detected (169, 170, 171).

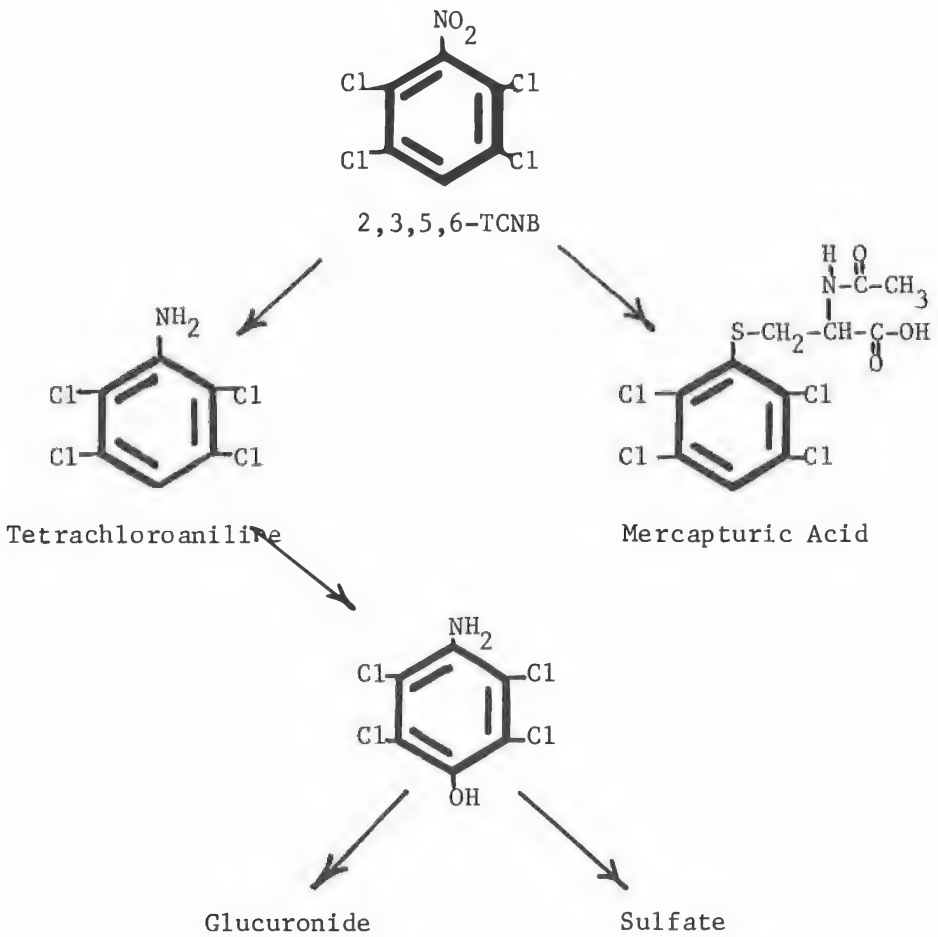


2,3,4,6-TCNB

After dosing rabbits with 2,3,4,6-TCNB, the corresponding aniline and N-acetyl-S-(2,3,4,6-tetrachlorophenyl)cysteine were found in the urine (111).



Some reduction of the nitro group took place in the gut after administration of 2,3,5,6-TCNB to rabbits. Very small amounts of tetrachloroaniline, mercapturic acid, free 4-amino-2,3,5,6-tetrachlorophenol, a sulfate and a glucuronide were excreted in the urine (169, 170, 171).



2,4,5,6-Tetrachloroisophthalonitrile (TCIN, Daconil)

When yeast cells were exposed to TCIN, the derivatives formed resembled those formed by the reaction of glutathione with TCIN *in vitro.* Coenzyme A and 2-mercaptoethanol also readily formed derivatives with TCIN *in vitro.* The nature of these derivatives was studied using 2-mercaptoethanol. Infrared spectra and chromatography of the four derivatives obtained indicated that one to four of the halogens had been substituted by 2-mercaptoethanol (1745).

No residues of daconil were found in millk of dairy cows fed this compound nor was intact daconil found in the manure or urine. Daconil rapidly decomposed in fresh rumen contents with production of two unidentified metabolites (586).

Tetramine [Tetramethylenedisulfotetramine]

Uniformly, C^{14}-labeled tetramine was administered to *Pseudotsuga menziesii, Dactylis glomerata* and *Rubus ursinus.* Tetramine moved upward in the xylem, but once deposited in tissue, became immobile. Slow degradation occurred in shoot tissue. Some $C^{14}O_2$ evolved (1185).

Thimet (Phorate) (0,0-Diethyl S-[ethylthio]methyl phosphorodithioate)

Thimet-P^{32} in oil was administered orally to male albino rats. Excretion in feces and urine varied with applied dosage. Hydrolysis products appearing in urine of the rats within 2 days were separated by ion exchange chromatography and found to consist of 0,0-diethyl phosphoric acid (17%); 0,0-diethyl phosphorothioic acid (80%); and 0,0-diethyl phosphorodithioic acid (3%). Using rat liver slice preparations, phosphorodithioate sulfoxides and sulfones and phosphorothiolate sulfoxides and sulfones were formed; and less than 1% of the radioactive metabolites appeared as hydrolysis products or unextractable residue from the proteins (151).

P^{32}-labeled thimet was orally administered to a lactating Holstein cow. Only hydrolysis products were present in the urine. Initially, 0,0-diethyl phosphorothioic acid was excreted. This declined as 0,0-diethyl phosphoric acid increased. Some 0,0-diethyl phosphorodithioic acid was also excreted. Similar findings were obtained with bile fluid. Fractionation and partitioning showed that the initial oxidation products in feces were the sulfoxide and/or sulfone. The highest total tissue residues appeared in liver, kidney, lung, alimentary canal, and glandular tissues. Fat samples were very low in thimet metabolites (151).

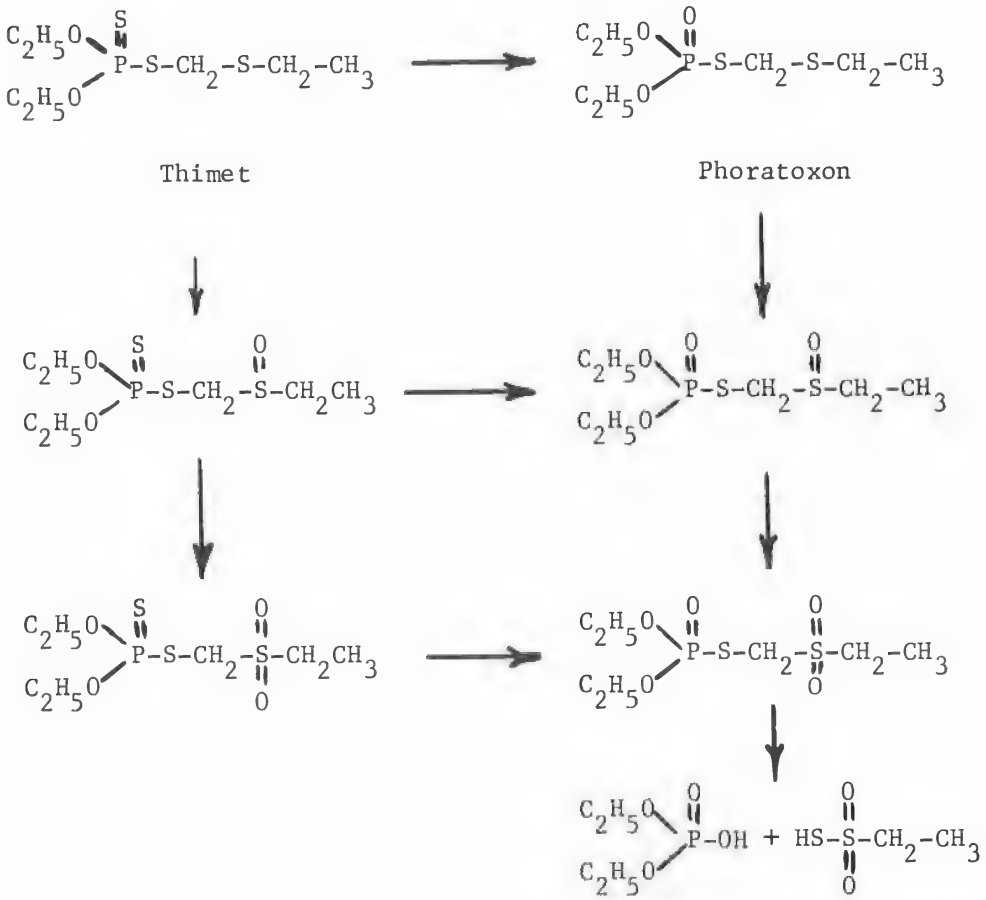
After thimet was applied to plants, total anticholinesterase activity increased for several days and there was an increase in the amount of material that is more polar in its solubility properties. However, some thimet persisted as such for as long as 32 days. By means of chromatography, infrared, and partitioning characteristics, four non-hydrolyzed metabolites were identified as the phosphorodithioate sulfoxide and sulfone, phoratoxon, phosphorothiolate sulfoxide and sulfone. 0,0-diethyl phosphorothioic and 0,0-diethyl phosphoric acid were also found (150, 151, 357, 992, 1519, 1774).

When labeled thimet was applied to soil, there was an initial loss by volatilization as great as 25% within one hour. After that, little or no volatilization occurred. A large portion of the remaining thimet was bound to the soil and could not be extracted with chloroform nor identified. As the amount of organic material present in the soil increased, the amount of thimet recovered decreased. Extraction, column chromatographic and partitioning techniques showed that soil applications of thimet were also partially oxidized and hydrolyzed (543).

After exposure of the yeast-like plant Torulopsis to thimet, the presence of the phosphorodithioate, phosphorothiolate, and other products of oxidation and hydrolysis were shown (98).

Thimet was applied topically to the German cockroach, large milkweed bug, and Rhodanus bugs. Analyses showed the sulfoxide and/or sulfone; but there was no evidence for the "oxon" pathway in these insects (977). When southern armyworm larvae were allowed to feed on red kidney bean plants grown in treated emulsions of thimet, results were similar. In addition to the sulfoxide and sulfone, 0,0-diethyl phosphorothioate and 0,0-diethyl phosphoric acid were found (151).

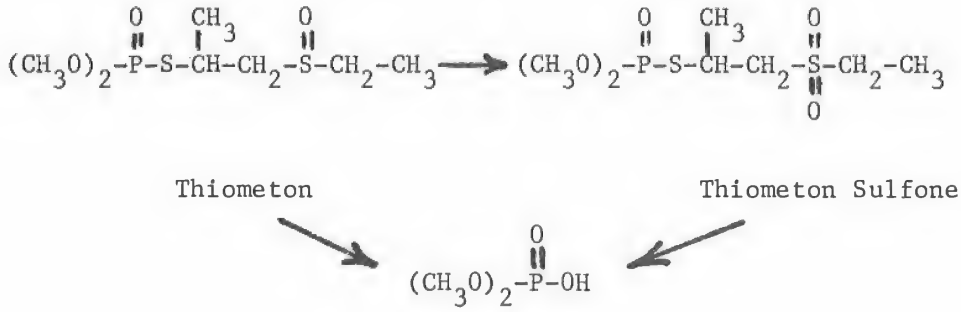
Exposure of thimet to ultraviolet light (maximum at 2540 A) produced the corresponding sulfone and sulfoxide (1608).



Thiometon (Bayer 23655) [O,O-Dimethyl S-isopropyl-2-sulfinylethyl phosphorothiolate]

Thiometon is similar to demeton S and its metabolism was also similar. Thiometon penetrated and translocated rapidly into citrus and rice plants. After penetration, thiometon underwent oxidization to the sulfone and hydrolysis to the phosphoric and dimethyl phosphoric acids (1414).

Under the influence of ultraviolet light (maximum of 2540 A), thiometon gave rise to the sulfone and sulfoxide (1608).



TIBA [2,3,5-Triiodobenzoic Acid]

In studies with rats, pigs and chickens, iodide, 2,5-diiodobenzoic acid and 3,5-diiodobenzoic acid were found in the organs (706, 1676). Two whole body retention components were observed--one with a biological half-life of 11.8 - 17.9 hours and the second 395 - 423 hours. Thyroid uptake was also indicated (706, 707).

When dairy cows were fed TIBA, no residues were found in the milk or feces. Some free TIBA and free and conjugated 2,5-diiodobenzoic acid were found in the urine. In steers fed TIBA, there were indications of urinary excretion of 2,5-diiodobenzoic acid and other partially de-iodinated benzoic acids. TIBA was stable when incubated with rumen fluid, thyroid or liver (581).

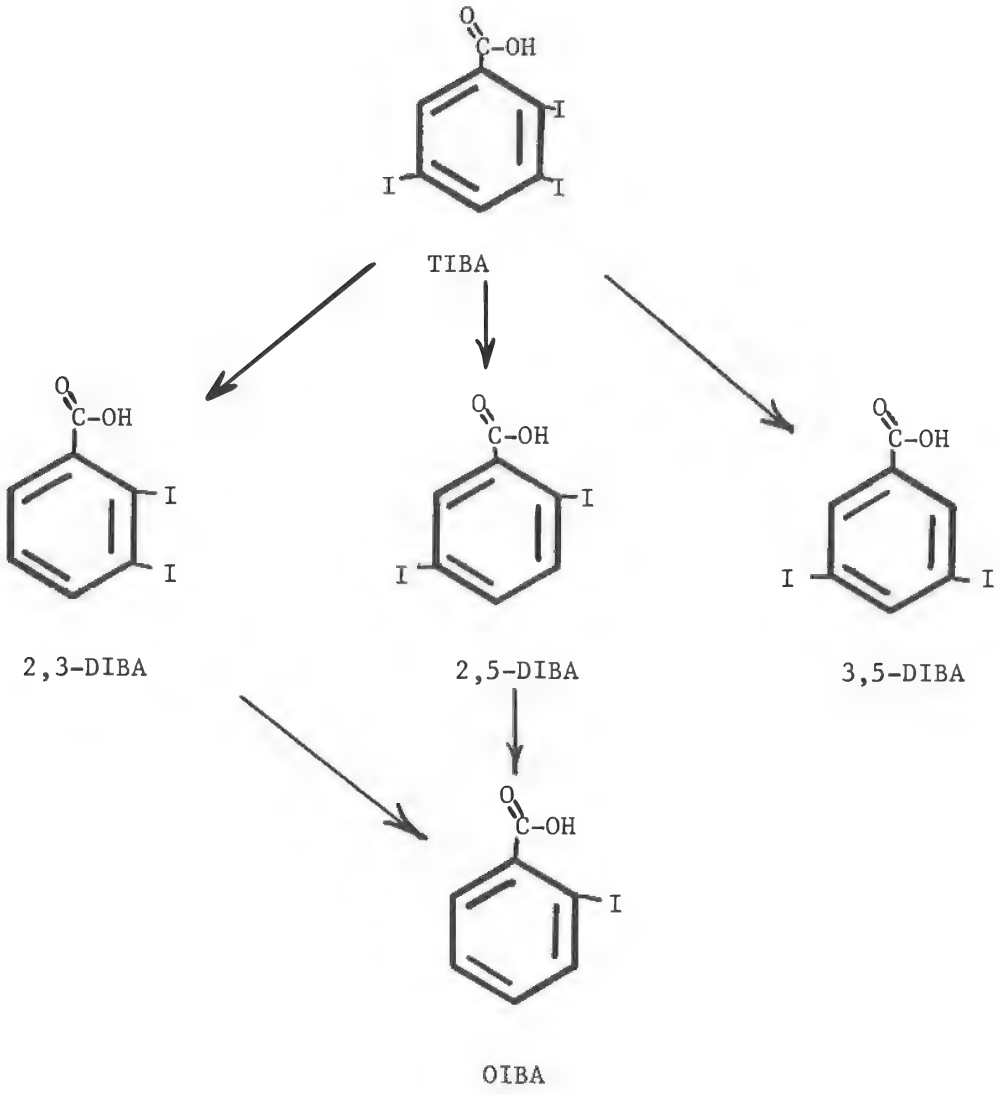
In lactating cows and goats, the primary route of excretion was via the kidney. Metabolic studies of the urine indicated TIBA and nine metabolites. Four of these were identified as 2,5-DIBA, 2,3-DIBA, OIBA and iodide ion. The remainder was thought to be conjugated forms of OIBA, TIBA, and DIBA. Residues of TIBA and/or its metabolites were distributed throughout all tissue samples analyzed with highest concentrations in thyroid and lowest in the brain (707).

Whole body retention studies indicated two biological half-life components represented by

$$Y = 19.9e^{(-0.0164)\ (X\ hr.)} + 71.8e^{(0.0512)\ (X\ hr.)}$$

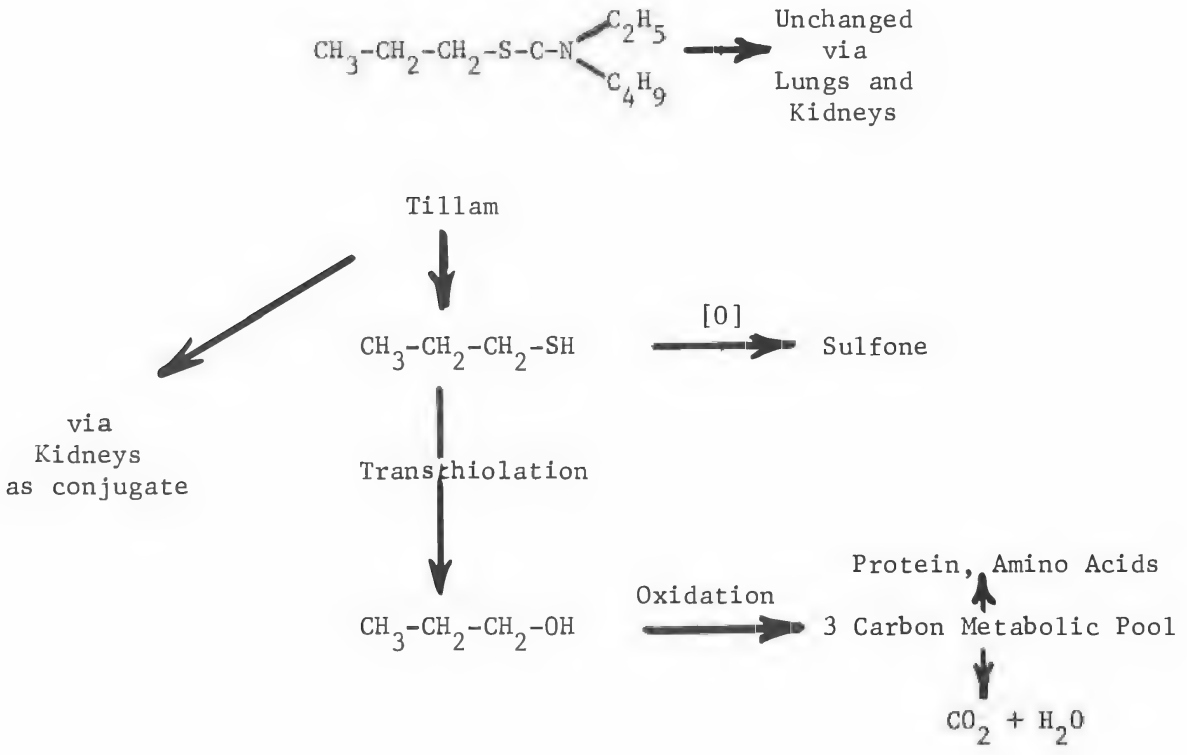
Where Y is the percent retained at "X" hours (707).

Exposure of C^{14}-carboxy labeled TIBA to UV gave rise to 2,5- and 3,5-diiodobenzoic acids (707).


TIBA
2,3-DIBA
2,5-DIBA
3,5-DIBA
OIBA

Tillam [S-Propyl-n-butylethylthiocarbamate]

Plant cells readily degrade Tillam-C^{14} and give rise to $C^{14}O_2$. In rats, tillam is rapidly eliminated. While excretion via the urine averages about 23%, feces samples were low, indicating that tillam is easily absorbed from the gastrointestinal tract and rapidly oxidized. Feeding studies with rats with labeled tillam gave rise to labeled amino acids, urea, and a conjugate which has not been identified (428, 429).



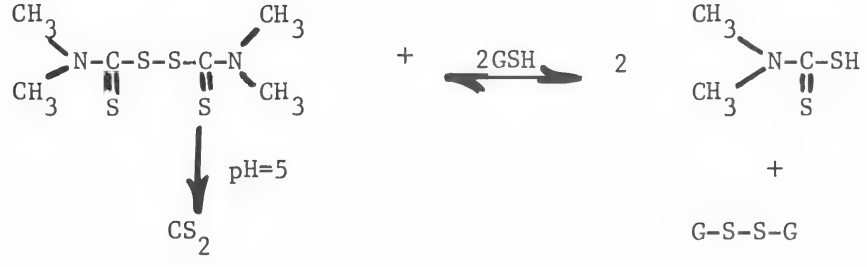
Tinox [0,0-Dimethyl O-(2-methylmercaptoethyl)phosphoro -thoate and -thiolate]

Studies concerning the metabolism of Tinox in tomato plants demonstrated the presence of both isomers as well as the oxidation metabolites of both isomers. Using P^{32}-tinox and TLC, extracts exhibited the presence of the thiono-sulfoxide and sulfone and the thiol-sulfoxide and sulfone (358).

TMTD (Tetramethyl thiuram disulfide, Thiram) [Bis(dimethylthiocarbamoyl) disulfide]

TMTD is known to inhibit fermentation of glucose, fructose-1,6-diphosphate, and glyceraldehyde-3-phosphate and to inhibit triosephosphate dehydrogenase (1311). TMTD reacted with thiols by a radical exchange mechanism. The final products are the corresponding disulfide of the parent thiol and dimethyldithiocarbamate (1123, 1274). (See also Dithiocarbamates and TTD.)

In studies with ruminant animals fed corn treated with TMTD, rumen microorganisms degraded TMTD to carbon disulfide and probably hydrogen sulfide and dimethylamine. Within six hours after dosage, 78% of the ingested TMTD was recovered as CS_2. Some unchanged TMTD was also observed in feces and urine (1221).



TOK [2,4-Dichloro-4'-nitrodiphenyl ether]

After feeding of 5 ppm TOK in forage to cows, no residues of TOK were found in the milk, urine, or feces. In the presence of fresh rumen fluid, a metabolite was produced which had a retention time, on gas chromatography, identical with that of 2,4-dichloro-4'-aminodiphenyl ether (587).

Toxaphene

Very little information is available about the metabolism of toxaphene. Using bioassay methods, it was found that resistant flies could metabolize 4.0 mcg of toxaphene from an absorbed dose of 5.4 mcg. No metabolites were identified.

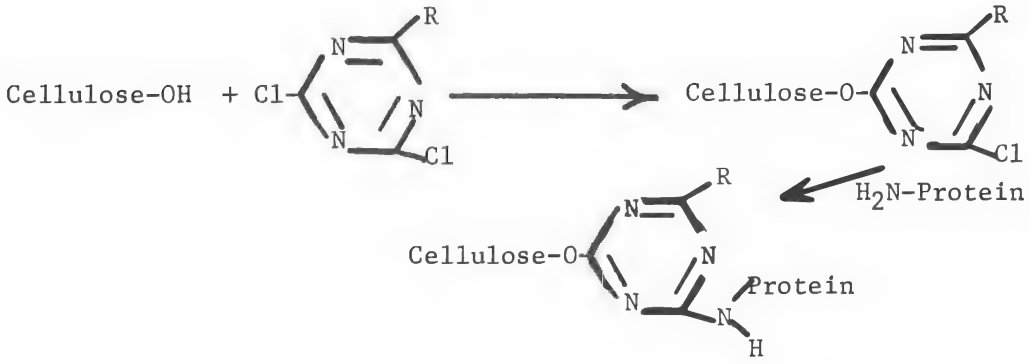
Toxaphene was stored unchanged in fatty tissue of treated animals. Infrared showed presence of all absorption bands exhibited by toxaphene. Comparative bioassay and total chlorine showed that total organic chloride in fatty tissue had about same toxicity to flies as an equivalent amount of toxaphene (368).

In studies with the cotton leafworm, the presence of a dehydrochlorinase and the breakdown of toxaphene was demonstrated. Three components were separated by paper chromatography but were not identified (398).

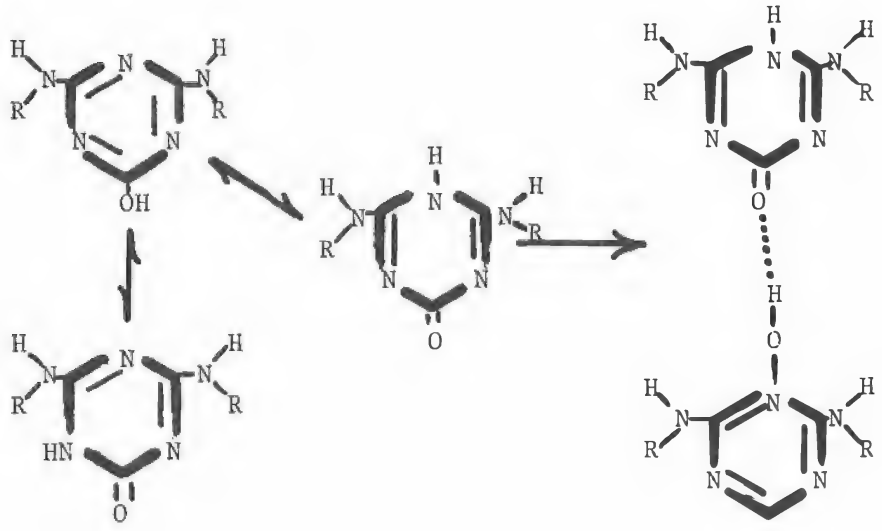
Triazines

Since chlorosis is the first sign of the effect of triazines on plants, interference with CO_2 assimilation and sugar formation can be expected. Studies showing that the Hill reaction is inhibited confirmed this.

Studies have shown that dichloro-s-triazines were capable of reacting with polysaccharides (cellulose) and proteins at or below room temperatures (54, 772, 1542).



Infrared spectra indicated that, in the dry state, triazines and their metabolites existed in a polymeric state (271).



Studies of the metabolism of triazine herbicides by mammals are few. In tests with a related triazine, cyanuric acid was excreted in the urine of mice fed triethylenimino-s-triazine (1066). In other studies, a labeled triazine analog was administered to male Swiss albino rats. Analysis of plasma and urine by paper chromatography showed that only the 6-hydroxy derivative was formed (28).

Adsorption of simazine and atrazine by soil fit the Freundlich equation $a=kc^n$, wherein

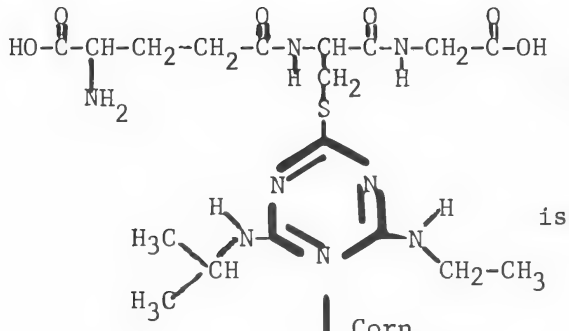
a = amount solute adsorbed

c = conc. of solution

k = constants for the

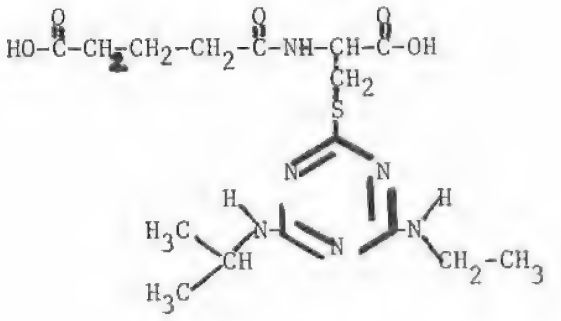
n = adsorbent and adsorbate

(1390).



(From Atrazine)
S-(4-ethylamino-6-isopropylamino-2-s-triazino) glutathione

Corn
Sorghum



γ-Glutamyl-S-4-ethylamino-6-isopropylamino-2-s-triazino)cysteine

Atrazine [2-Chloro-4-ethylamino-6-isopropylamino-s-triazine]

Feeding studies with atrazine in rats indicated 20 metabolites (73). In other feeding studies with rats and rabbits, dealkylation was observed. Metabolites were isolated and identified (See table 1). In addition to the tabulated metabolites, rabbits also excreted N-(2-chloro-4-amino-s-triazinyl-6)glucoside (133). In a goat and sheep, the ring in labeled atrazine was not metabolized; the side chain was metabolized (72).

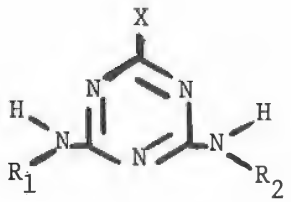


Table 1

Metabolites

	X=				
	Cl			OCH_3	SCH_3
	Simazine	Atrazine	Propazine	Prometone	Prometryn
$R_1=R_2=H$	+	+	+	+	+
$R_1=H$, $R_2=C_2H_5$	+	+	-	-	-
$R_1=H$, $R_1=i\text{-}C_3H_7$	-	+	+	+	+
$R_1=H$, $R_2=CH_2COOH$	+	+	-	-	-
$R_1=H$, $R_2=CH(CH_3)COOH$	-	+	+	+	-
Parent Compounds					
R_1	C_2H_5	C_2H_5	$i\text{-}C_3H_7$	$i\text{-}C_3H_7$	$i\text{-}C_3H_7$
R_2	C_2H_5	$i\text{-}C_3H_7$	$i\text{-}C_3H_7$	$i\text{-}C_3H_7$	$i\text{-}C_3H_7$

C^{14}-Atrazine was applied to susceptible and resistant plants. These studies showed that all test plants converted some atrazine to hydroxyatrazine and that the amount was related somewhat to resistance (493, 1079, 1080, 1224). Degradation of C^{14}-ring-labeled atrazine to $C^{14}O_2$ was not a significant process in plants (349). Hydroxyatrazine was also metabolized readily by oats (493).

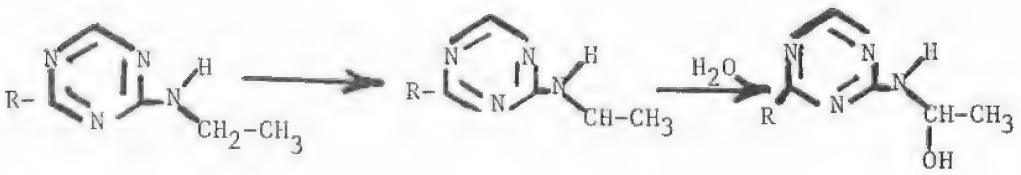
The metabolism of atrazine was investigated in resistant corn (Zea mays L.) and sorghum (Sorghum vulgare Pers.), in intermediately susceptible pea (Pisum sativum L.), and in highly susceptible wheat (Triticum vulgare Vill.) and soybean (Glycine max Meril.). Results of these studies showed that two pathways for atrazine metabolism existed. All higher plants studied were able to metabolize atrazine by N-dealkylation of either of the two alkylamine groups. In addition to this, those plants containing benzoxazinone (Corn and wheat) were also able to detoxify atrazine by hydrolysis in the 2-position and formation of hydroxyatrazine (1292, 1293, 1294, 1295, 1297). In corn, atrazine metabolism yielded the desethyl 2-hydroxy and desisopropyl 2-hydroxy atrazine analogs as well as the 2-hydroxyatrazine (1748). In the leaves of corn and sorghum, two sulfur compounds have also been found and identified as: S-(4-ethylamino-6-isopropylamino-2-s-triazino)glutathione and γ-glutamyl-S-(4-ethylamino-6-isopropylamino-2-s-triazino)cysteine. Both compounds form rapidly in leaf discs incubated with atrazine. An enzyme system has been isolated from corn and sorghum leaves which catalyzes the formation of metabolite from atrazine and glutathione (1759). The hydroxyatrazine was also observed in cotton (Gossypium hirsutum L.) (122).

Hydroxyatrazine was detected as a degradation product after perfusion of soils with atrazine (32, 614, 615, 616). Hydrolysis was apparently largely responsible for the degradation. The rate of hydrolysis to form hydroxyatrazine in aqueous systems, or in systems containing sterilized soil, was first-order (31). Side chain and ring cleavage occurred in solution culture studies (1771). On montmorillonite, chemical degradation occurred. NMR and infrared studies showed that the hydrolytic degradation product was predominantly the keto form of the protonated hydroxy analog (1590).

$C^{14}O_2$ production from ring-labeled atrazine by microorganisms has been observed (7, 349). The fungus, F. roseum, metabolized atrazine to hydroxyatrazine (309). Penicillium cyclopium, P. frequentans, P. purpurogenum, Aspergillus niger, A. repens, Ceyhalosporium acre monium and Cladosporium herbarum also decomposed atrazine (984).

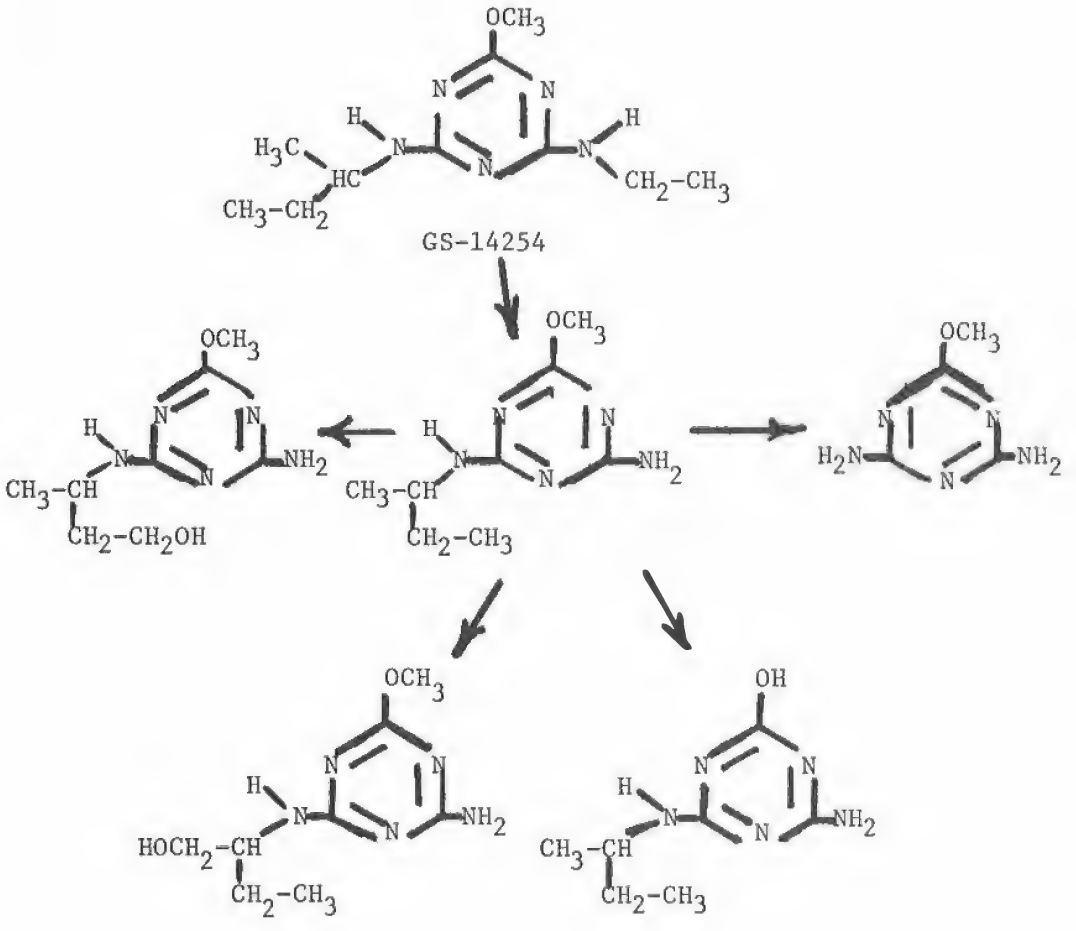
With Fenton's reagent, atrazine underwent side chain hydroxylation (1676).

$$H_2O_2 + Fe^{++} + H^+ \longrightarrow HO^{\cdot} + H_2O + Fe^{+++}$$



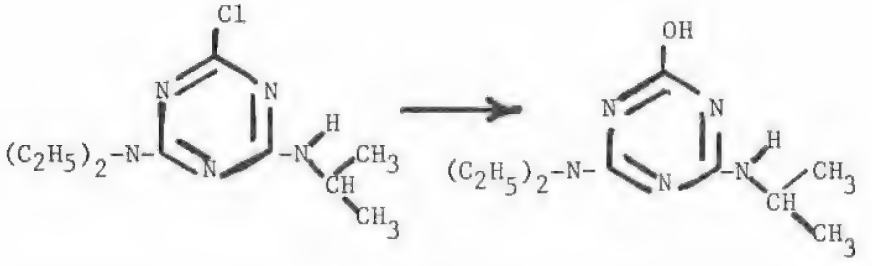
2-Methoxy-4-sec-butylamino-6-ethylamino-s-triazine (GS-14 254)

After a cow was given the methoxy-labeled triazine, about 15% of the label was expired. When ring-labeled material was administered, 76% of the dose was found in the urine and 10% in the feces after 120 hours. When methoxy-labeled triazine was administered to a goat, 83% of the dose appeared in the urine after 48 hours and 3% in the expired air after 24 hours. Urinary metabolites were the same in both animals. Sixteen metabolites were observed in urine and feces. Those identified were: 6-amino-4-sec-butylamino-2-methoxy- ; 4,6-diamino-2-methoxy- ; 6-amino-4-(1-hydroxy-sec-butylamino)-2-methoxy- ; 6-amino-4-(4-hydroxy-sec-butylamino)-2-methoxy- ; and 6-amino-4-sec-butylamino-2-hydroxy-s-triazines (1756).



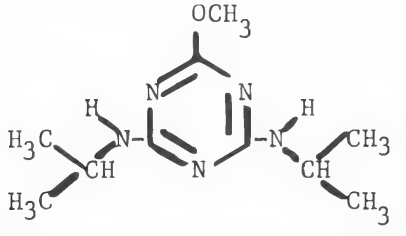
Ipazine [2-Chloro-4-diethylamino-6-isopropylamino-s-triazine]

Cotton did not extensively degrade the triazine ring of C^{14}-labeled ipazine. Evolution of $C^{14}O_2$ from culture solutions was not observed; however, $C^{14}O_2$ evolution when the soil was treated, indicated the role of soil microorganisms in ipazine degradation. Some hydroxyipazine was found but this did not constitute the major degradation product (607).



Prometone [2-Methoxy-4,6-bis(isopropylamino)-s-triazine]

Feeding studies in rats have indicated eleven metabolites (73). Some metabolites were isolated and identified in feeding studies with rats and rabbits (See Table 1) (133).

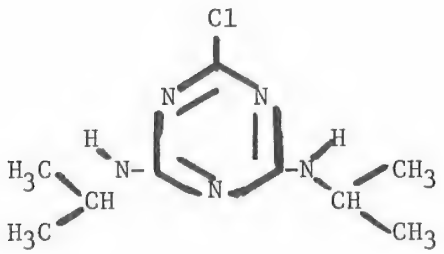


Propazine [2-Chloro-4,6-bis[isopropylamino]-s-triazine]

Feeding studies with rats indicated oxidation of the side chains to CO_2 and eighteen metabolites (73). In other studies with rats and rabbits, some metabolites were isolated and identified (See Table1) (133). After propazine was administered to lactating goats, sixteen metabolites were separated by ion-exchange chromatography of the urine but not identified. CO_2 was obtained from dealkylation and oxidation of the side chain isopropyl moieties (1633).

Propazine-C^{14} was metabolized by cane (Sorghum vulgare L.) but not oats (Avena)(475, 476, 477).

In soil samples treated with propazine, accumulation of the hydroxy derivative indicated initial hydrolysis at the 2-position (616).



Prometryne [2-Methylmercapto-4,6-bis(isopropylamino)-s-triazine]

Rats and rabbits fed prometryne excreted 2-mercapto-4-amino-6-isopropylamino-s-triazine and bis(2-amino-4-isopropylamino-s-triazinyl-6-yl) disulfide. The mono- and di- dealkylated prometryne analogs were also observed (133). See Table 1.

When cotton plants were treated with C^{14}-labeled prometryne, labeled material accumulated in roots, stems, leaves and lysigenous glands but no $C^{14}O_2$ was detected (1496).

Eight day old pea plants were exposed to prometryne and harvested eight days later. Several metabolic pathways were indicated. In one, the side chain of prometryn was metabolized and 2-hydroxy-4-amino-6-isopropyl-amino-s-triazine was identified. In the other, the methylthio group was oxidized to the sulfone which readily hydrolyzes to the hydroxy analog. Chromatography indicated the presence of the sulfoxide and hydroxy analogs (1165).

Studies with E. coli showed that prometryne could replace thymine or uracil and was incorporated into nucleic acids (1394). Some microorganisms appeared to degrade prometryne by oxidation of the methylmercapto group to a sulfoxide or sulfone (593). It was found that some fungi also degrade prometryne: Penicillium cyclopium, P. frequentans, P. purpurogenum, Aspergillus niger, A. repens, Ceyhalosporium acre monium and Cladosporium herbarum (1003).

Simazine [2-Chloro-4,6-bis(ethylamino)-s-triazine]

In feeding studies with rats and rabbits, dealkylation was observed (See Table 1) (133).

Work with labeled simazine has shown that a wide variety of plants, resistant and nonresistant, metabolize this material (473, 477, 1286). Using ring-labeled simazine-C^{14}, it was found that field corn (Zea mays) produced little (about 2%) $C^{14}O_2$ (347, 348, 477) whereas in sweet corn the rate of decomposition to $C^{14}O_2$ was high (1188).

Other studies have demonstrated the formation in plants of 2-hydroxy-4,6-bis(ethylamino)-s-triazine from simazine (253, 254, 270. 509, 593, 594, 595, 606, 1024, 1025, 1026, 1027, 1028, 1165, 1240). This reaction occurred in light or dark (1163).

A cyclic hydroxamate was postulated as the responsible agent in the metabolism of triazines by plants and a mechanism was proposed. This was confirmed with the isolation of 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one from Zea mays and the demonstration that it can convert simazine to its hydroxy analog (250, 253, 603, 605, 655, 687, 1241, 1451).

The tolerance of several species of Gramineae was studied. Hydroxysimazine and several other unidentified metabolites were observed. Tolerance was not related to the ability of their excised roots to metabolize C^{14}-simazine; however, the content of benzoxazinone derivatives was directly related to the ability of excised roots to form hydroxysimazine and another minor metabolite. Roots not containing benzoxazinone derivatives did not form hydroxysimazine but other minor metabolites were detected (604).

Studies have shown that many soil microorganisms metabolize simazine (436, 573, 574, 767, 769, 1030, 1172, 1188). One of the major metabolites has been identified as hydroxysimazine (614, 615). In some organisms, degradation proceeded via dealkylation, deamination, or both, and hydroxysimazine was not an intermediate. A major metabolite has been identified as 2-chloro-4-amino-6-ethylamino-s-triazine (778).

C^{14}-ring-labeled simazine was fed to Coix lacryma L. After ethanol extraction and fractionation on a cation exchange column, the radioactive metabolites were 2-hydroxy-4,6-diethylamino-s-triazine and 2-hydroxy-4,6-diamino-s-triazine (703).

Although the path is not known, it has also been shown that some soil microorganisms can open the triazine ring and degrade it to CO_2 (573, 574, 1030, 1172, 1188). Fungi metabolized the side chains but did not open the ring to any appreciable extent (309, 770).

A number of fungi capable of degrading simazine were isolated and identified (143, 767, 1003).

Aspergillus flavipes (Bainier and Sartory)

Aspergillus fumigatus Fres.

Aspergillus ustus (Bainier)

Aspergillus niger

Aspergillus repens

Fusarium moniliforme Sheld

Fusarium oxysporum Schlecht

Penicillum purpurogenum Stoll sp.

Penicillium cyclopium

Penicillium frequentans

Stachybotrys sp.

Trichoderma viride Pers. exFr.

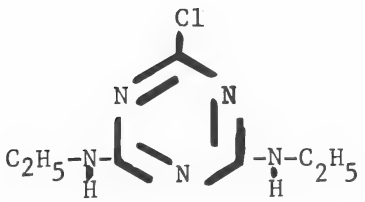
Ceyhalosporium acre monium

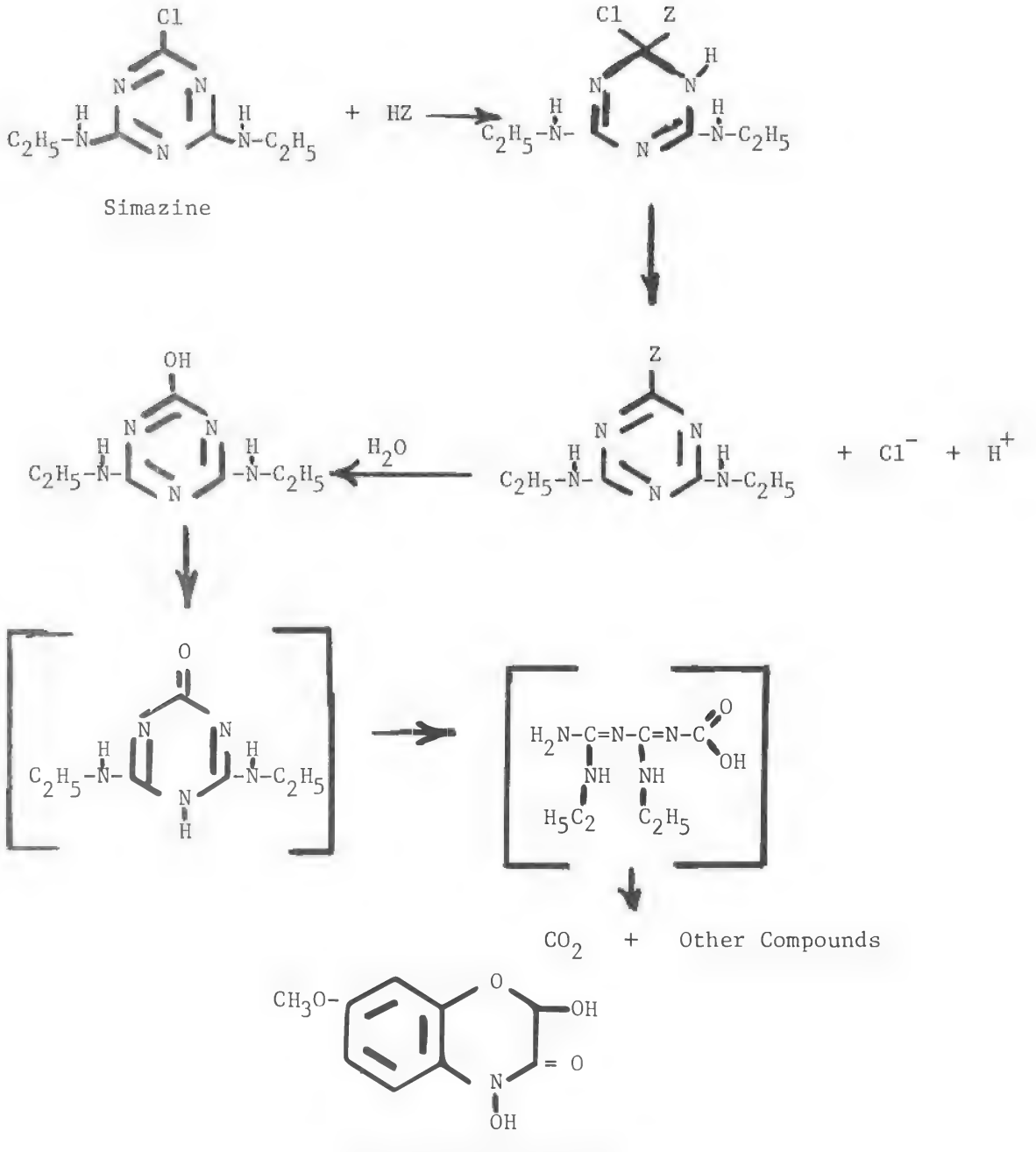
Cladosporium herbarum

Pseudomonas: red-orange and blue green

Streptomyces: three unidentified

Four believed to be Arthrobacter





(Z = Cyclic Hydroxamate)

2,4-Dihydroxy-7-methoxy-1,4-benzoxazin-3-one

Trichlorfon (Dipterex, Bayer L 13/59) [0,0-Dimethyl 2,2,2-Trichloro-1-Hydroxyethyl Phosphonate]

^{32}P-labeled trichlorfon was administered to a cow. Analysis of blood indicated a rapid hydrolysis. From urine samples, trichloroethanol glucuronic acid was isolated and identified. Dimethylphosphate and desmethyl trichlorfon were found in the milk (33, 1219, 1669). In piglets, DDVP appeared in the blood after subcutaneous injection of trichlorfon (1787). With in vitro studies with serum of cows, at 37°C, trichlorfon was degraded to DDVP, desmethyl trichlorfon and dimethylphosphate (1696).

In rat brain homogenates, C^{14}-labeled trichlorfon was rapidly metabolized to monodemethylated trichlorfon, monomethylphosphate, 2,2,2-trichloro-1-hydroxyethyl phosphonic acid, and a fourth unidentified compound (628). Following injection in rats of trichlorfon labeled in the two methyl groups, 60% of the C^{14}, was recovered after 24 hours in the expired air and urine. $C^{14}O_2$ and C^{14}-formate constituted about 50% of the recovered radioactivity (627). Additional studies were conducted with P^{32}-labeled trichlorfon. After interperitoneal injection, 75-85% of the administered radioactivity appeared in the urine within 48 hours. Three P^{32}-labeled compounds were found in the urine. Two were identified as mono- and dimethylphosphates. The third metabolite was not identified (632).

After exposure of larvae of the cotton leaf worm (Prodenia lituria F.) to labeled trichlorfon, 0-methyl-2,2,2-trichloro-1-hydroxyethyl phosphonic acid, 2,2,2-trichloro-1-hydroxyethyl phosphonic glucuronic acid (65-75%), mono- and dimethylphosphate, and $C^{14}O_2$ (629, 1536) were found.

In other studies, after exposure of adult larvae of Prodenia litura F. to P^{32} labeled trichlorfon, the hemolymph contained 3 times more radioactivity than in the gut. While the concentration in the gut and hemolymph decreased with time, there was an accumulation of radioactivity in the fat. The metabolites excreted were identified as monomethyl- and dimethylphosphates, and the glucuronate of the demethylated trichlorfon (1537).

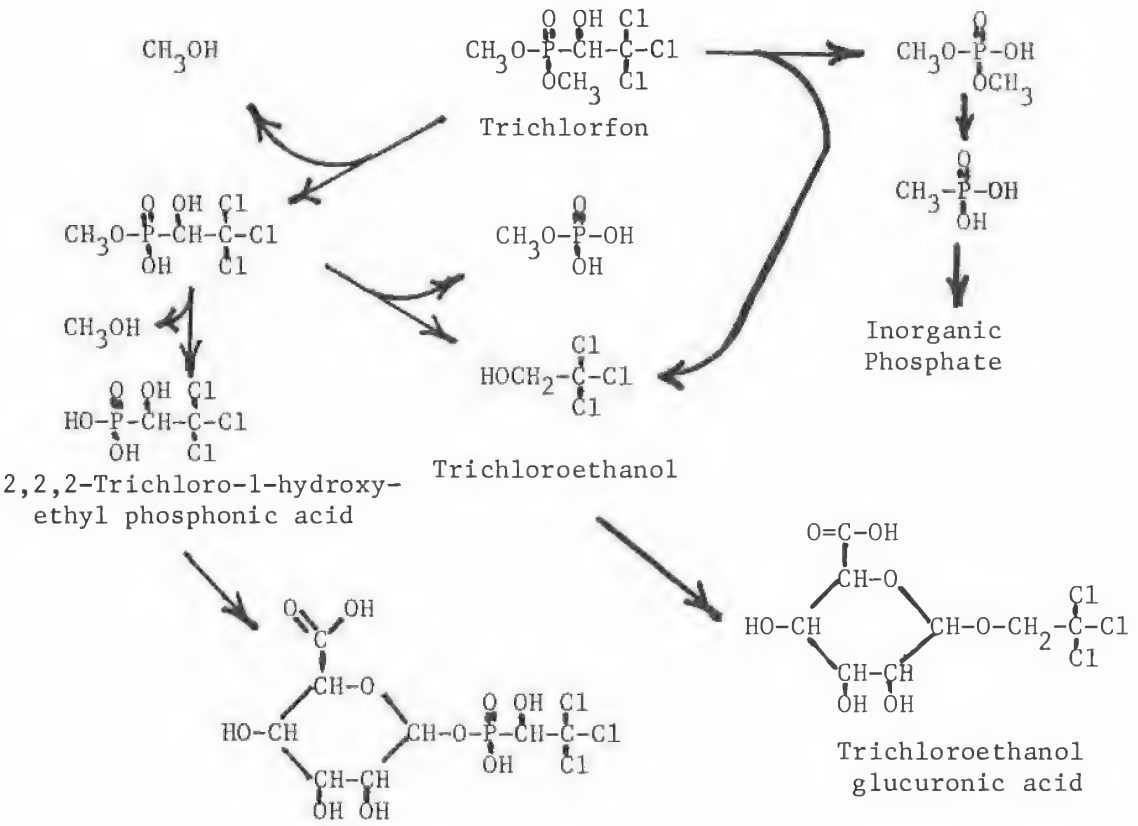
When trichlorfon was mixed with digestive juices or blood of silkworm larvae (Bombyx mori L.), trichlorfon disappeared and DDVP appeared. At present however, it is not certain whether the rapid degradation of trichlorfon in the digestive juice (pH 10-11) was enzymatic or not. At room temperature in 0.1N NaOh solution, trichlorfon gave rise to several compounds, one of which was identified as DDVP (1595).

When cotton plants were exposed to P^{32}-labeled trichlorfon, dimethylphosphate, monomethylphosphate, and inorganic phosphate were found. Some formaldehyde formed and give rise to CO_2 (633, 1044).

Tests with microorganisms showed that Aspergillus niger, Pencillium notatum, and Fusarium sp. hydrolyzed trichlorfon. One compound was identified as O-methyl-2,2,2-trichloro-1-hydroxyethyl phosphonic acid. The other is believed to be 2,2,2-trichloro-1-hydroxyethyl phosphonic acid (1537, 1539).

In acidic aqueous media, trichlorfon was hydrolyzed to dimethyl phosphate plus trichloroethanol and to methanol and desmethyl trichlorfon. At pH 5.0 and greater, DDVP formed spontaneously (993, 1015, 1696).

(See also Butonate and DDVP).

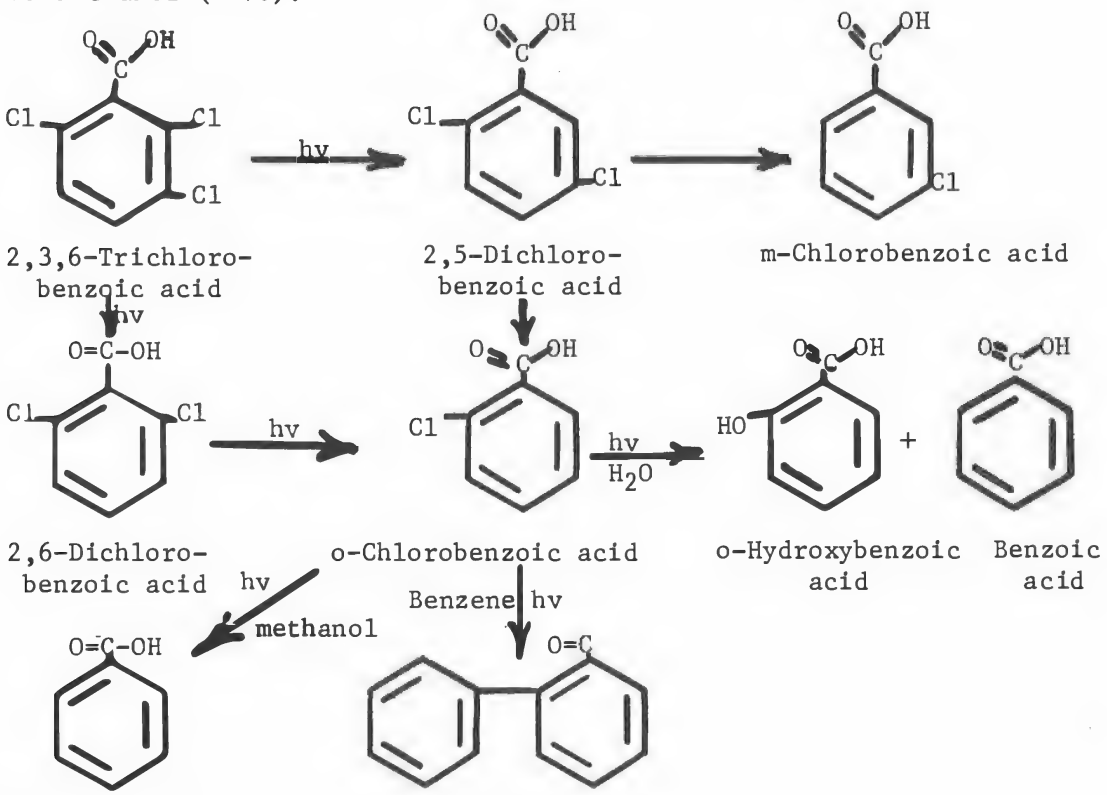


2,3,6-Trichlorobenzoic Acid

Compared to most herbicides, 2,3,6-trichlorobenzoic acid has been found to be much more stable in soil. Studies with biologically active soil showed that this material was degraded with release of inorganic chloride. No intermediates were identified (367, 1158).

When fed to rabbits and mice, 17.6% and 2.7%, respectively, of the 2,3,6-trichlorobenzoic acid fed was excreted (74). Studies with other benzoic acids have shown that, when fed to animals, they are excreted in urine either unchanged or as conjugates of glycine and glucuronic acid (1089, 1508).

Under the influence of ultraviolet light, TBA underwent dechlorination to both 2,5- and 2,6- dichlorobenzoic acid. Further dechlorination produced o-and m-chlorobenzoic acid. Further exposure of the ortho isomer to ultraviolet light in aqueous media produced benzoic acid and o-hydroxybenzoic acid; in benzene, o-biphenylcarboxylic acid was produced. Ortho- and meta- chlorobenzoic acid were both dechlorinated to benzoic acid in methanol (1170).



Trifluralin [α,α,α-Trifluoro-2,6-dinitro-N,N-di-n-propyl-p-toluidine]

Excretion of radioactivity by rats and dogs was virtually complete within three days following administration of a single oral dose of $C^{14}F_3$-trifluralin--78% appeared in the feces and 22% in the urine. In addition to unchanged trifluralin, a mono amino analog (VI) was found in the feces. Three metabolites were identified in the urine. One compound (IV) was formed by complete dealkylation; the second (III), by loss of a propyl group and reduction of one nitro group; the third (V), by loss of both propyl groups and reduction of one nitro group. The probably intermediate (II), formed by loss of one propyl group, was observed by gas chromatography but never isolated because of the small amounts present. All other compounds were isolated. Melting points were obtained for all solids and matched with authentic material. Infrared, TLC, and X-ray diffraction were also used to identify metabolites (405).

In vivo studies were conducted with a cow and a goat. The major product identified from both urine and feces was compound VII (649).

In vitro degradation of trifluralin in artificial rumen fluid was studied. Use of TLC, GC, and radiochemical procedures revealed a rapid destruction of trifluralin. The principal products formed were: N^2, N^2-di-n-propyl-3-nitro-5-trifluoromethyl phenylenediamine (VI) and N^4, N^4-di-n-propyl-α,α,α, -trifluorotoluene-3,4,5-triamine (VII) (553).

Carrots were grown from seeds in trifluralin treated soil for 110 days and then harvested. Thin-layer chromatography and radioautography of carrot root indicated that α,α,α-trifluoro-2,6-dinitro-N-n-propyl-p-toluidine (II) was the most prominent metabolite. α,α,α-Trifluoro-2-amino-6-nitro-N-n-propyl-p-toluidine (III) and 3,5-dinitro-4-di-n-propyl-aminobenzoic acid (X) were also detected (553).

The metabolism of trifluralin was studied in autoclaved sand by intact peanut (Arachis hypogaea L.) and sweet potato (Ipomoea batalas L.) plants and crude extracts of these plants. Both intact plants and crude extracts degraded trifluralin. With crude extracts of peanuts, the initial degradation product was an incompletely dealkylated product (II). This was converted to compound III by reduction of one nitro group. Degradation also proceeded by reduction of one nitro group to compound VI with subsequent dealkylation to compound III. Compounds VII and IX were also observed. Two other metabolites were detected but not completely characterized: compound XI, a phenol; and compound XII, a dealkylated benzoic acid. Degradation of trifluralin by sweet potato extracts followed similar paths; however, the initial degradation apparently was the reduced compound VI. Compounds XI and XII were also observed (1772).

Experiments with C^{14}-trifluralin revealed different degradative pathways under aerobic and anaerobic soil condition. Aerobically, degradation proceeds through dealkylation and progressive reduction. Anaerobically, reduction is followed by dealkylation (1182).

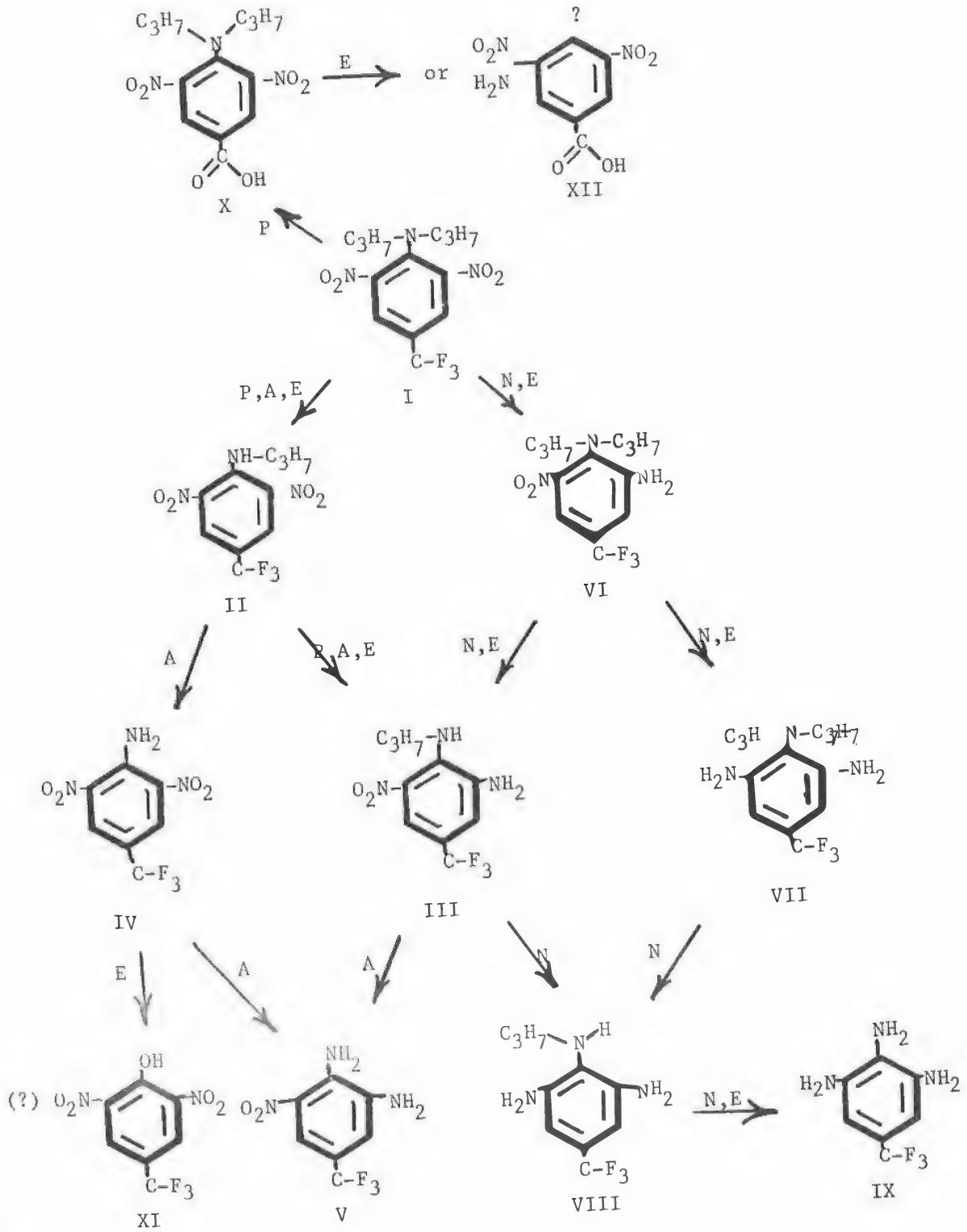
Aerobic degradation of trifluralin in soil gave rise to small amounts of compounds II, III, IV, V, and CO_2. An unresolved mixture of polar products, probably condensed aromatic amines, was also present. Under anaerobic conditions in soil, compounds III, VI, VII, VIII and IX were observed. A scheme of the postulated metabolic pathways was indicated (553, 1183).

Rumen bacteria degraded trifluralin via dealkylation (compound II) and reduction of the nitro group (compound VI). Further reduction of compound II and dealkylation of compound VI gave rise to metabolite III. The N-propylamino-diamino metabolite (VIII) was observed. This could have arisen from metabolite III and/or by dealkylation of metabolite VII, which was also found. Other unidentified stable metabolites were also observed (1757).

Studies with trifluralin-exposed Aspergillus niger revealed unchanged trifluralin and a small amount of the mono propyl analog; with Sclerotium rolfsii Fusaruim sp., and Trichoderma sp., revealed only unchanged trifluralin (512). Data from studies with Lachnospira multiparus and bacteroides ruminicola subsp. brevis suggested that these organisms alter trifluralin with cleavage of a propyl group and reduction of a nitro group to an amine (1682).

Pure trifluralin was coated on glass and soil surfaces and exposed to sunlight and to ultraviolet light having a wavelength range of 300-700 millimicorons. Progressive change occurred with time and intensity of exposure. The end-product after six hours' exposure to sunlight was red-brown in color. Exposure of trifluralin to 4,000 ft-c of light from a mercury-vapor source for 4 hours produced similar results. No products were identified (1602).

When dilute heptane solutions of trifluralin in borosilicate glass or quartz vials were exposed to ultraviolet light, extensive decomposition occurred. The photodecomposition products have not been identified. However, the product isolated from N-propyl-2,6-dinitro-4-trifluoromethyl-aniline was identified as 2-nitro-6-nitroso-4-trifluoromethylaniline (928).


E
or
?
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XII
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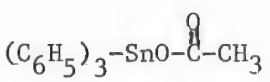
P = Plants
A = Aerobic
N = Anaerobic
E = Plant Extracts

Triphenyl Tin Acetate



Triphenyl tin acetate was poorly absorbed by plants. When taken in by animals, probably as a residue on the green fodder, this compound was rapidly excreted with the dung. The small amount absorbed was partly excreted in the urine and partly distributed throughout the whole body. The active ingredient has a biological half-life of 20 hours while its metabolic products, presumed to be bound inorganic tin, have a biological half-life of 8 to 70 days (650).

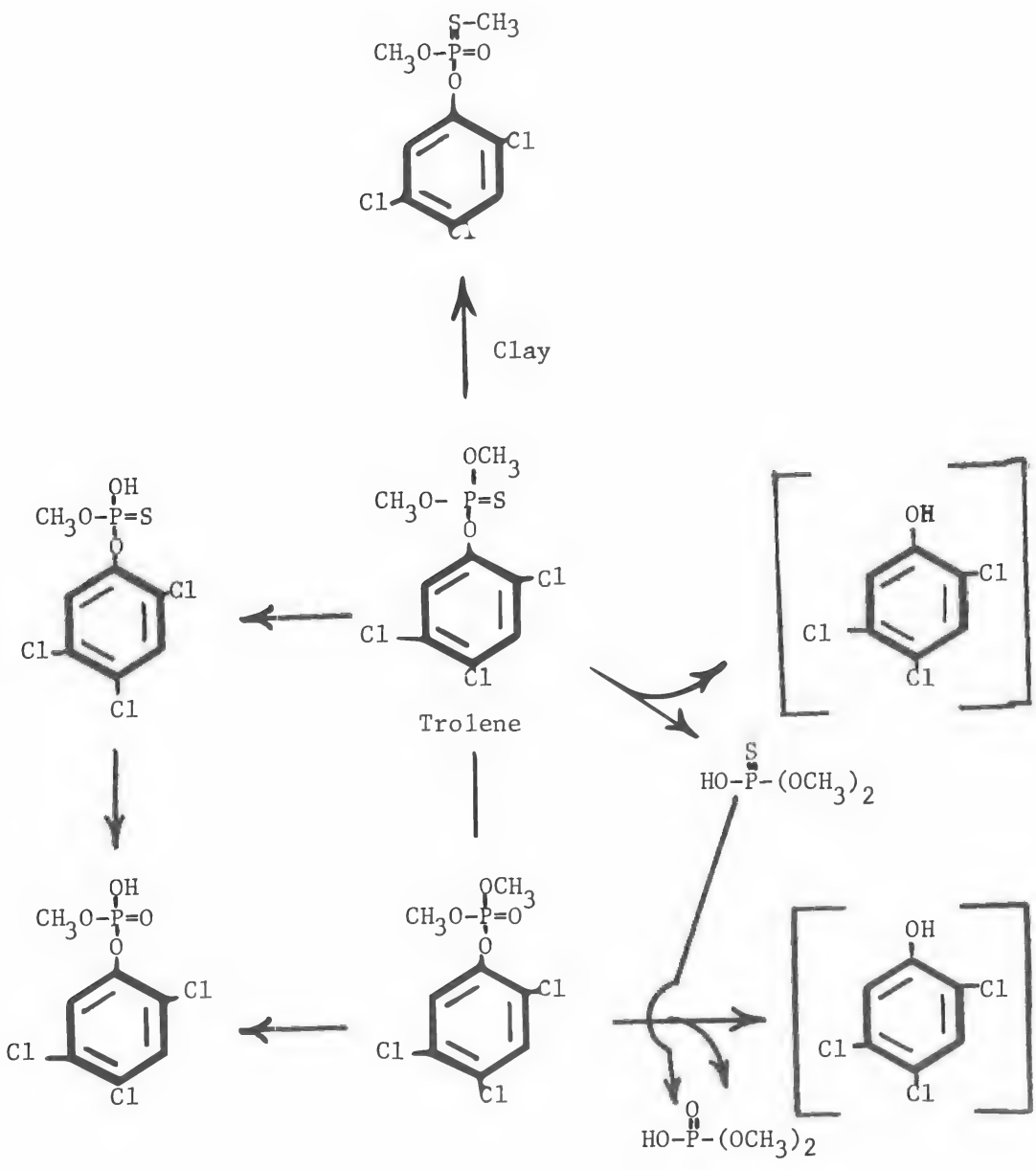
Trolene (Ronnel) [0,0-Dimethyl 0-(2,4,5-trichlorophenyl)phosphorothioate]

After administration of radioactive trolene, the highest levels of trolene and its derivatives in all tissues were reached after 12 hours. Equilibration was reached after about 12 days. Residues persisting 7 days after oral administration were similar to trolene and its oxygen analog. The tissues in order of decreasing persistence of trolene and metabolites were those of: liver, kidney, spleen, subcutaneous and mesenteric fat, heart, brain.

Trolene and its oxygen analog were hydrolyzed in rats and excreted in the urine initially as phenyl phosphoric and phosphorothioic acids. These decreased progressively while excretion of dimethyl phosphoric acid increased.

When labeled trolene was fed to cows, the highest levels appeared in the fat, kidneys, and lungs. Fractionation of brain phospholipids showed some radioactivity in the lecithin-cephalin and sphingomyelin fractions. The peak level in the urine occurred from 18 to 32 hours after treatment. The predominant metabolite in the urine was identified as 0-methyl-0-hydrogen 0-(2,4,5-trichlorophenyl) phosphorothioate. In rumen fluid, trolene was hydrolyzed to yield phenyl phosphoric acids. Some dimethyl phosphoric and phosphorothioic acids were also produced. Application of trolene to houseflies showed that the primary site of hydrolysis was at the phosphorus-oxygen-methyl bond as in rumen fluid (1167).

Decomposition of trolene is normally base-catalyzed. When clays treated at elevated temperatures were used as carriers, a new type of decomposition occurred. After treatment at 950°C, all OH^- is gone and the clay becomes a mixture of oxides. The decomposition product becomes one of molecular rearrangement (1238).


Clay
Trolene
Inorganic
Phosphate
Phospholipids
and
Phosphoproteins

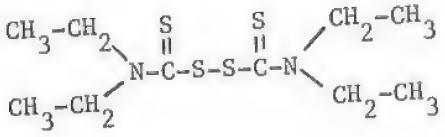
TTD (Disulfiram) [Tetraethylthiuram disulfide]

Plant tissues transformed N,N-dialkyldithiocarbamates into the corresponding glucosides (760) and ananine derivatives (761). By contrast, microorganisms produced aminobutyric acid derivatives (1304).

In animals, enzymes converted TTD to diethyldithiocarbamate, which in turn decomposed to CS_2 (457, 741, 1179). In man CS_2 was expired for 4-5 hours after treatment and about 50% of the administered TTD was recovered as CS_2 (987). Ninety percent of an S^{35}-labeled dose was absorbed from the intestinal tract and 10% was excreted in the feces. Analysis of urine indicated that about 65% of the sulfur was oxidized to sulfate and about 6% was recovered in the organic sulfur fraction (400). Excretion of unchanged TTD in urine is practically nil (373, 875); however, small amounts of both changed and reduced TTD were recovered from urine (400). The reduced form, diethyldithiocarbamic acid, has been detected in blood corpuscles and in alkaline urine but not in plasma (875). Recently, N,N-diethylthiocarbamoyl-1-thio-β-glucopyranosiduronic acid was isolated from the combined urine of four men given oral doses of TTD. Identification followed paper chromatography, isolation, infrared analysis and comparison of the melting points of the isolated and synthetic triacetyl methyl esters (759).

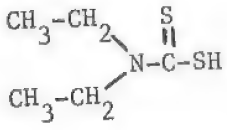
Using S^{35}-labeled material, a significant amount of the S^{35} was found attached to the serum proteins and to the soluble proteins of liver (1369). The greater part of this radioactivity was liberated by the addition of glutathione and the product was identified as diethyl dithiocarbamate (1368, 1370). *In vitro* studies also showed that TTD reacted with glutathione (740).

Using rats, S^{35}-labeled TTD was administered intraperitoneally. The unchanged material was never detected in plasma, liver, or urine. Sulfate, CS_2, the S-glucuronide of diethyldithiocarbamate, and traces of the free thiol were found in the urine. In addition to these metabolites, there were protein bound mixed disulfides in the plasma and liver (1369, 1370).



TTD

↓



Diethyldithiocarbamic
Acid

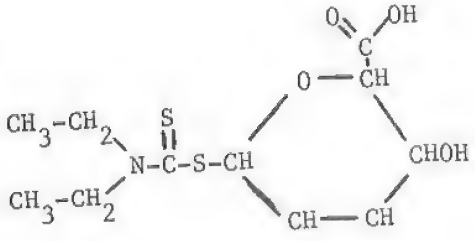
→ $(CH_3-CH_2)_2-NH + CS_2 \uparrow$

$+ H_2S$

↓

$SO_4^{=}$

↓



N,N-Diethylthiocarbamoyl
1-Thio-β-D-Glucopyranosiduronic
Acid

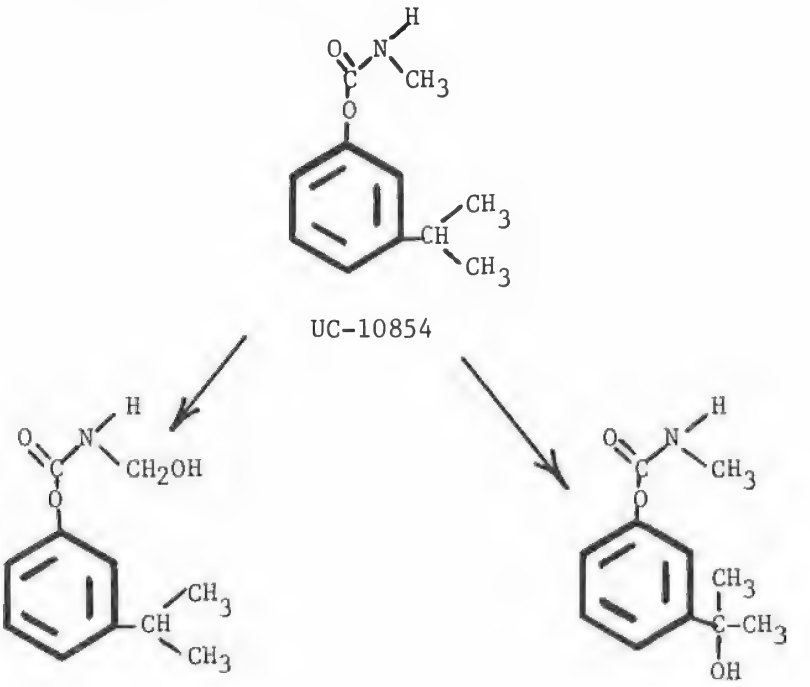
UC-10854 [m-Isopropylphenyl N-methylcarbamate] (m-Cumenyl methylcarbamate)

When rats were exposed to labeled UC-10854, labeled $C^{14}O_2$ was formed (836). After incubation of rat liver microsomes with UC-10854 in the presence of reduced nicotinamide—adenine dinucleotide phosphate (NADPH), 3-(1-hydroxy-1-methylethyl)phenyl methylcarbamate and 3-isopropyl N-hydroxymethylcarbamate were observed (1110, 1112). Several unidentified compounds were also found (1111).

Studies conducted with UC-10854 labeled N-$C^{14}H_3$ and C^{14}=O showed that $C^{14}O_2$ arose from both in treated houseflies. Houseflies also hydrolyzed this material to the corresponding phenol (1001).

After treatment of bean plants with UC-10854, in addition to several unidentified compounds, the hydroxypropyl and N-hydroxymethyl analogs were found (3, 845, 846).

(See also CEPC, CIPC and IPC).

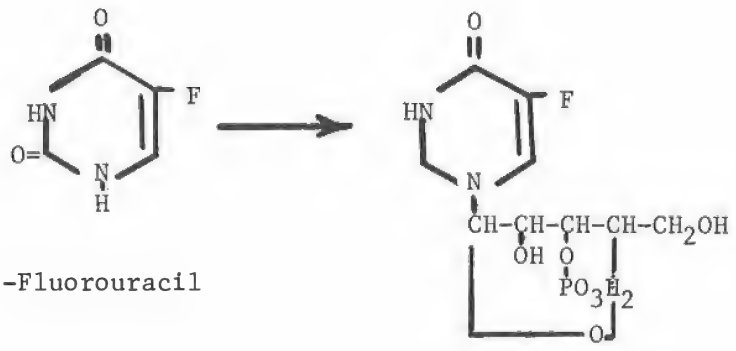


Uracil Herbicides

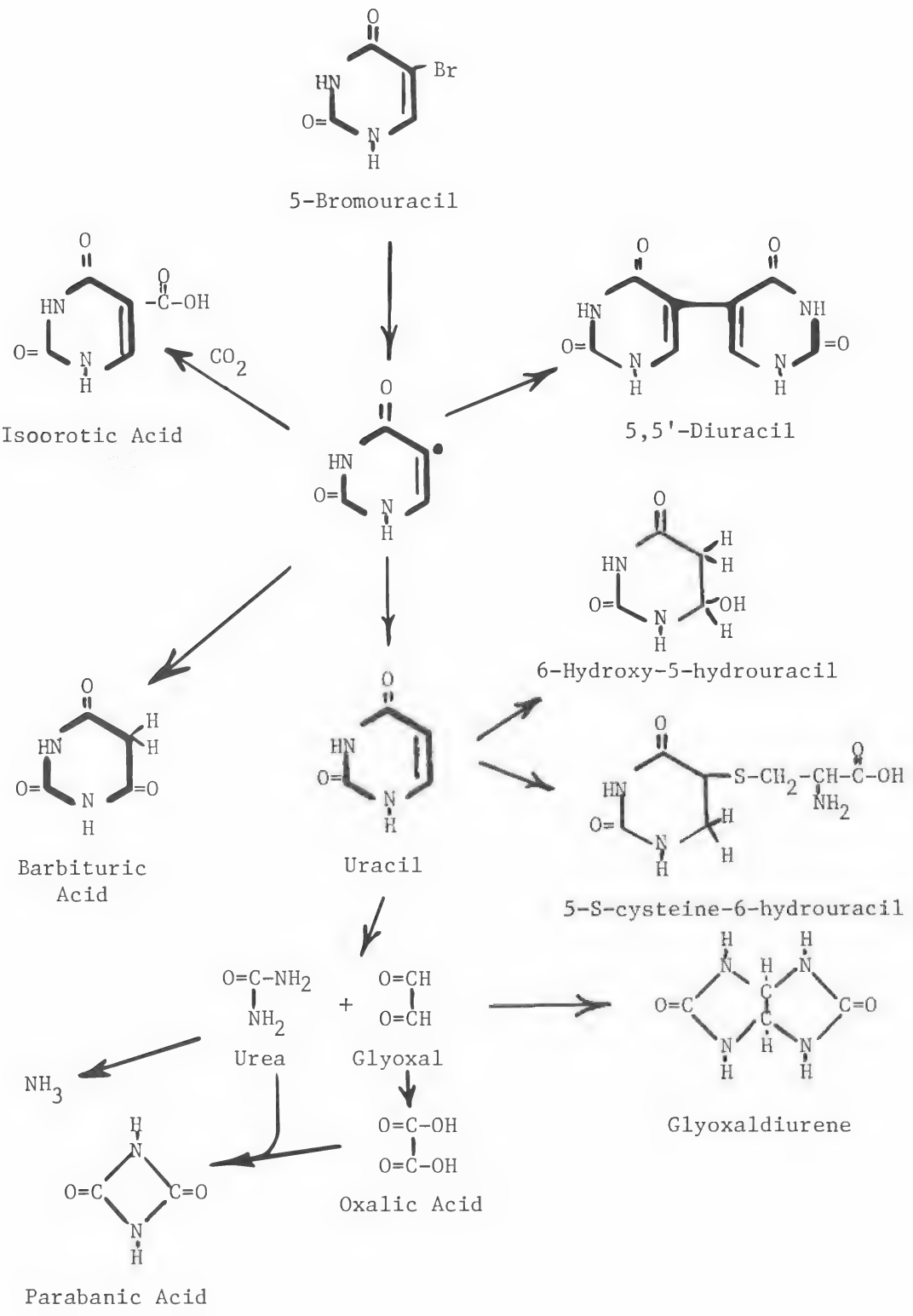
Uracil herbicides do not interfere with pyrimidine metabolism. Instead they appear to act as strong inhibitors of the photochemical reaction of isolated chloroplasts [the Hill reaction] (664).

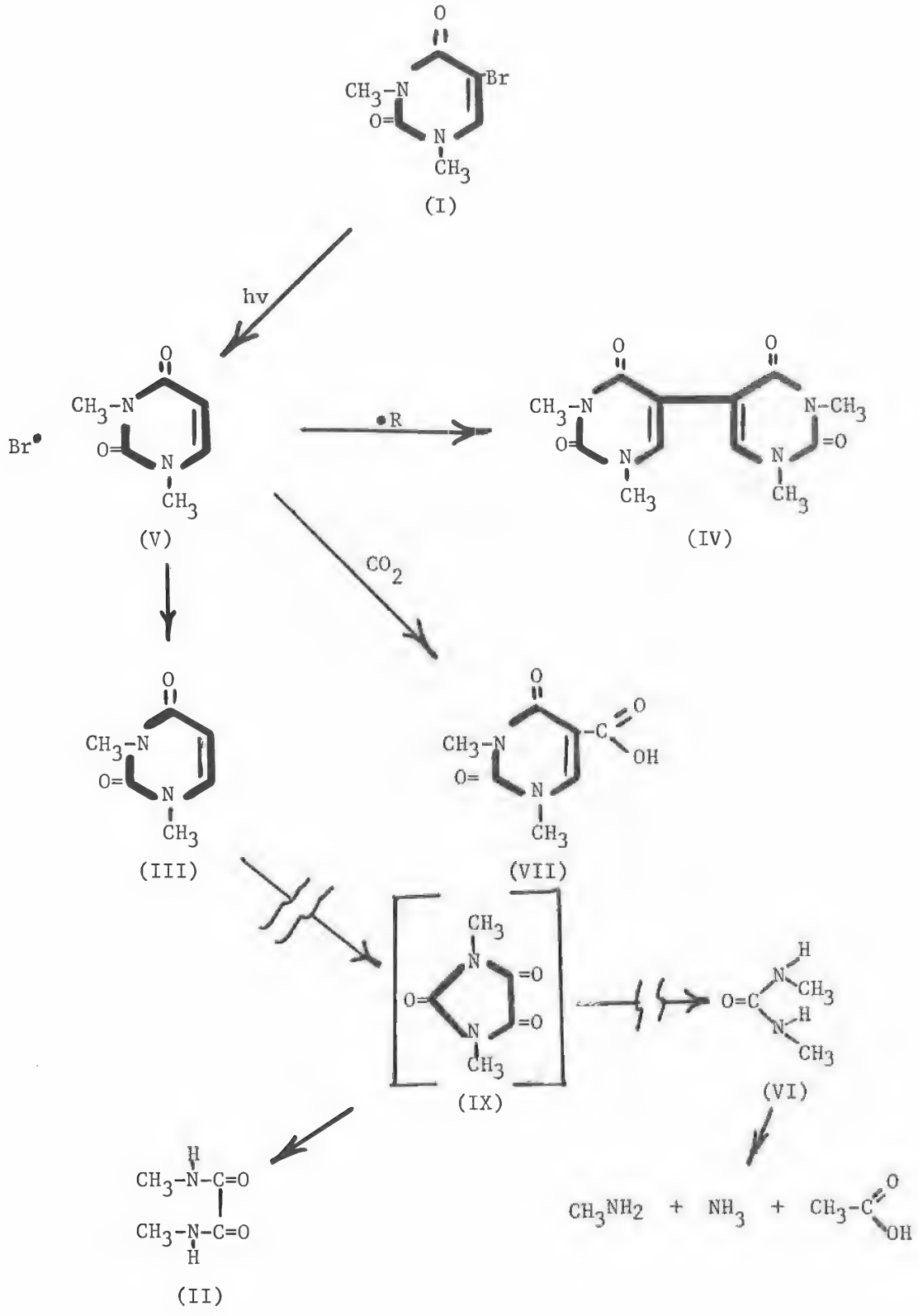
In an effort to understand the manner in which the uracils act as inhibitors of photochemical reactions, several substituted uracils were exposed to ultraviolet irradiation. When 5-bromo-1,3-dimethyluracil (I) in aqueous solution was irradiated with a G.E. germicidal tube (G 15T8 which emits mainly at 254 mμ), sym-dimethyloxamide (II), 1,3-dimethyluracil (III), and 5,5'-di-1,3-dimethyluracil (IV), sym-dimethylurea (VI), 5-carboxy-1,3-dimethyluracil (VII), methylamine, ammonia, and acetic acid were isolated and identified (713, 715). Studies showed that 5-bromouracil underwent similar photochemical changes even with biological doses of ultraviolet. 5,5'-Diuracil, uracil, glyoxaldiurene, barbituric acid, oxalic acid, isoorotic acid, parabanic acid, urea, ammonia, and glyoxal were formed. These studies showed that the photochemical process was a free radical reaction (714, 715). Irradiation of acidic uracil-cysteine solutions gave rise to a heterodimer identified as 5-S-cysteine-6-hydrouracil. In the absence of cysteine, 6-hydroxy-5-hydrouracil was formed (1333).

5-Fluorouracil, a chemosterilant, was incorporated into fly egg RNA as 5-fluorouridylic acid (792).



5-Fluorouracil


5-Bromouracil
Isoorotic Acid
CO_2
5,5'-Diuracil
6-Hydroxy-5-hydrouracil
Barbituric Acid
Uracil
5-S-cysteine-6-hydrouracil
Urea
Glyoxal
NH_3
Glyoxaldiurene
Oxalic Acid
Parabanic Acid


(I)
hv
Br•
•R
(V)
(IV)
CO2
(III)
(VII)
(IX)
(VI)
(II)
CH3NH2 + NH3 + CH3-C(=O)OH

Ureas

Ureas were inactivated by the action of ultraviolet light (743, 744, 1482, 1483) and by reaction with FMN in plants (1379). After exposure of N'-(4-chlorophenoxy)-phenyl-N,N-dimethylurea to ultraviolet radiations, thin-layer chromatography revealed small amounts of the mono- and demethylated derivatives (532).

Degradation by soil microorganisms apparently appeared to be the primary cause for disappearance of the urea herbicides (656, 1205). Some studies showed that ureas were demethylated step-wise and degraded to the aniline derivative (143, 333, 532, 1170).

A microsomal mixed function oxidase, which successively demethylates N,N-dimethylphenylurea herbicides, was isolated from etiolated cotton seedling hypocotyl and leaves of cotton, plantain, buckwheat, wild buckwheat and broadbeans. The enzyme requires NADPH or NADH and oxygen. Optimum pH is 7.4 to 7.8. The N-demethylated substrate and formaldehyde were identified by TLC and mass spectra. Diuron, fluometuron, monuron and monomethylmonuron were studied (1758).

Diuron [3-(3,4-dichlorophenyl)-1,1-dimethylurea]

In rats and dogs fed diuron, excretion of metabolites occurred in both feces and urine. N-(3,4-dichlorophenyl)urea was predominant in urine but N-(3,4-dichlorophenyl)-N'-methylurea, 3,4-dichloroaniline and 3,4-dichlorophenol were also detected (674).

After feeding diuron to albino rats, N-(3,4-dichlorophenyl)urea, N-(3,4-dichlorophenyl)-N'-methylurea and 3,4-dichloroaniline were found in the free state. 2-Acetamido-4,5-dichlorophenol, 6-acetamido-2,3-dichlorophenyl, and N-(2-hydroxy-3,4-dichlorophenyl)-N'-methylurea were found only in the bound form. 2-Amino-4,5-dichlorophenol, N-(2-hydroxy-4,5-dichlorophenyl)urea, N-(2-hydroxy-4,5-dichlorophenyl)-N'-methylurea, and N-(2-hydroxy-3,4-dichlorophenyl)urea were found free and bound as glucuronides or sulfates (134).

Root uptake of C^{14}-diuron from solution was studied. The major component in cotton leaves was identified as 1-(3,4-dichlorophenyl)urea. In soybean leaves, the major component was identified as 1-(3,4-dichlorophenyl)-3-methylurea. Small amounts of the monomethyl and demethylated derivatives were found in the nutrient solutions from cotton and soybeans as well as in oat and corn tops (1289, 1332). When leaf discs of cotton (Gossypuim hirsutum L.) and broadleaf plantain (Plantago major L.) were incubated with diuron, mono- and di- demethylated diuron and 3,4-dichlorophenylurea were observed. With soybean (Glycine max Merrill), only the mono- demethylated diuron was found. Degradation of diuron by corn (Zea mays L.) resulted only in minute amounts of the mono- demethylated diuron (1713).

Labeled diuron was applied to the leaf and to the nutrient solution of corn seedlings. Analyses of samples of leaf washes, nutrient, solutions and corn seedlings indicated the presence of 3,4-dichloronitrobenzene, 3-(3,4-dichlorophenyl)-1-methylurea, N-(3,4-dichlorophenyl)urea and 3,4-dichloroaniline (1109, 1586).

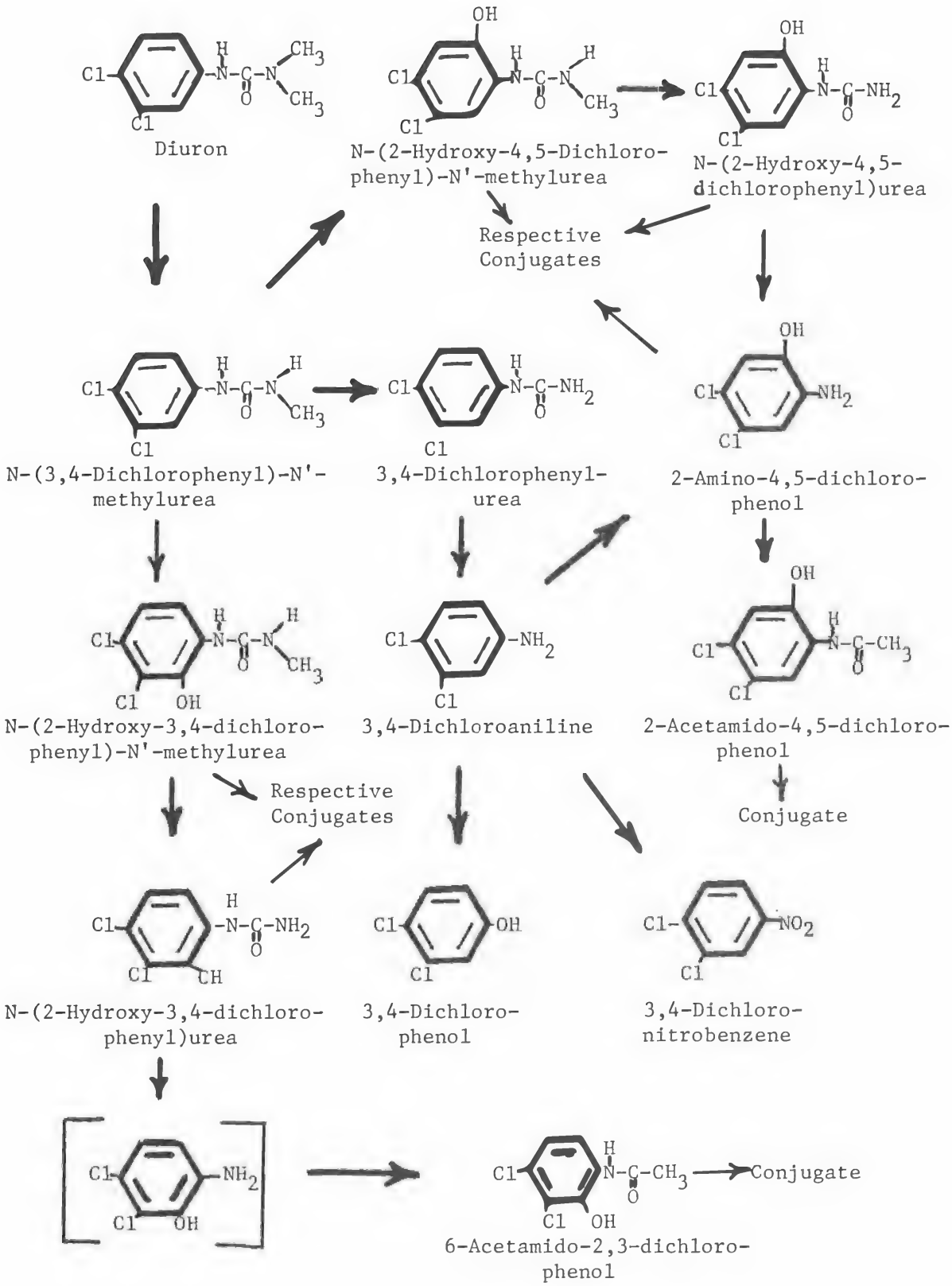
When oat seedlings were grown in a modified Lakeland sandy loam soil, an increase in soil pH resulted in greater amounts of diuron in the shoots. Herbicide content of roots was independent of pH, organic matter, or temperature modifications. In addition to diuron, mono demethylated diuron was observed in the shoots (1677).

Under field conditions, diuron degradation in soil was demonstrated by isolation and identification of intermediate breakdown products. After isolation and TLC, infrared spectra were used to confirm the identification of the degradation products (334): 3-(3,4-dichlorophenyl)-1-methylurea; 3-(3,4-dichlorophenyl)urea; 3,4-dichloroaniline (1289, 1728).

Fluometuron (Cotoran) [3-(m-trifluoromethylphenyl)-1,1-dimethylurea]

C^{14}-labeled fluometuron was metabolized in vivo, primarily by the liver, to the N-desmethyl and N-demethylated analogs which were excreted as conjugates in the urine. A small amount was excreted as the aniline analog. No labeled CO_2 was detected during the first 48 hours (154).

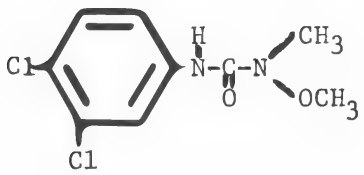
After treatment of cucumbers and cotton with fluometuron, the C^{14}-metabolites were determined by autoradiography. The monomethylated, demethylated, and aniline derivatives were identified from cotton. Only the mono- and de-methylated derivatives were identified from cucumber (1231, 1232, 1588).


Diuron
N-(2-Hydroxy-4,5-Dichlorophenyl)-N'-methylurea
N-(2-Hydroxy-4,5-dichlorophenyl)urea
Respective Conjugates
N-(3,4-Dichlorophenyl)-N'-methylurea
3,4-Dichlorophenylurea
2-Amino-4,5-dichlorophenol
N-(2-Hydroxy-3,4-dichlorophenyl)-N'-methylurea
3,4-Dichloroaniline
2-Acetamido-4,5-dichlorophenol
Respective Conjugates
Conjugate
N-(2-Hydroxy-3,4-dichlorophenyl)urea
3,4-Dichlorophenol
3,4-Dichloronitrobenzene
6-Acetamido-2,3-dichlorophenol
Conjugate

Linuron (Afalon) [N-(2,4-dichlorophenyl-N'-methyl-N'-methoxyurea]

Linuron in oil was fed to albino rats. Urine was collected and analyzed for metabolites (N-(3,4-dichlorophenyl)urea, N-(3,4-dichlorophenyl)-N'-methylurea and 3,4-dichloroaniline were found free. N-(2-hydroxy-4,5-dichlorophenyl)-N'-methylurea, N-(5-hydroxy-3,4-dichlorophenyl) urea and 6-acetamido-2,3-dichlorophenol were present as glucuronides or sulfates. Found bound and free were 2-amino-4,5-dichlorophenol, 2-acetamido-4,5-dichlorophenol, N-(2-hydroxy-4,5-dichlorophenyl)urea and N-(2-hydroxy-3,4-dichlorophenyl)urea (134).

Linuron was also degraded by plants giving the 3,4-dichloroaniline, and CO_2 (1549).



Monolinuron (Aresin) [N-(4-chlorophenyl)-N'-methyl-N-methoxyurea]

When aresin was fed to albino rats, the following metabolites were found both free and bound as glucuronides or sulfates: N-(2-hydroxy-4-chlorophenyl)-N'-methylurea, N-(3-hydroxy-4-chlorophenyl)-urea; traces of N-(4-chlorophenyl)-N'-methoxyurea, 2-amino-5-chlorophenol, 2-acetamino-5-chlorophenol, N,N'-bis-(4-chlorophenyl)urea, 2-acetamino-6-chlorophenol, and N-(2-hydroxy-4-chlorophenyl)-N'-methyl-N'-methoxyurea. N-(4-chlorophenyl)urea and a trace of 4-chloroaniline were found free only. N-(2-hydroxy-4-chlorophenyl)-N'-methylurea was found in the bound state only (417).

In plants, monolinuron was degraded to 4-chloroaniline and CO_2 (1549).

Monuron [N-(4-chlorophenyl)-N',N'-dimethylurea]

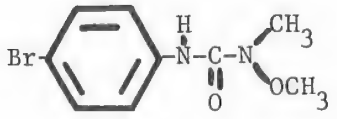
After oral administration of monuron in oil to albino rats, collected urine was analyzed for metabolites. N-(2-hydroxy-4-chlorophenyl)urea, N-(3-hydroxy-4-chlorophenyl)urea, N-(2-hydroxy-4-chlorophenyl)-N'-methyl-urea, and N-(4-chlorophenyl)-N'-methylurea were found free and bound as glucuronides or sulfates. N-(4-chlorophenyl)urea was observed only in the free state. Traces of N-(2-hydroxy-4-chlorophenyl)-N',N'-dimethylurea and 2-acetamino-5-chlorophenol were observed (417). In cotton, the monomethyl and the demethylated analogs as well as 4-chloroaniline were observed (1289).

When C^{14} monuron was used, 1-(p-chlorophenyl) urea and 1-(p-chlorophenyl)-3-methylurea were the major metabolites found in the leaves of cotton and soybeans, respectively. p-Chloroaniline was also found in cotton leaves but not in soybean leaves (1332). When leaf discs of cotton (Gossypium hirsutum L.), broadleaf plantain (Plantago major L.) and soybeans (Glycine max Merrill) were incubated with monuron, mono- and di-demethylated monuron and p-chloroaniline were observed. With corn (Zea mays L.), only the mono-demethylated monuron was observed (1713). An amidohydrolase has been obtained from soybeans and cotton leaf tissue that is capable of hydrolyzing monuron (561, 1715).

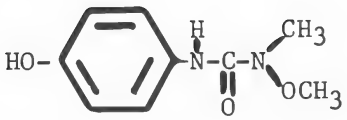
Monuron was degraded when solutions of this material were exposed to ultraviolet light. The major product found was the N-methyl-N-formyl analog. Other compounds were also observed: mono- and di- demethylated monuron, N-(4-chloro-2-hydroxyphenyl)-N',N'-dimethylurea, N-formyl-4-chloroaniline, 1,3-bis (p-chlorophenyl)urea and N-(4-chlorophenyl)-N'-formylurea (1639).

Patoran (Metobromuron) [3-p-Bromophenyl)-1-methoxy-1-methylurea]

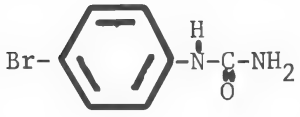
Photolysis of patoran was conducted with aqueous solutions exposed to sunlight for a 17-day period in late summer and by using a low pressure 2.5 watt mercury immersion lamp. Exposure of the aqueous solution to sunlight resulted in a mixture containing 80% unchanged patoran and 15% 3-(p-hydroxyphenyl)-1-methoxy-1-methylurea. The remaining material consisted of demethylated patoran, p-bromophenylurea and some unidentified materials. Some HBr was also probably formed (1634).



Patoran



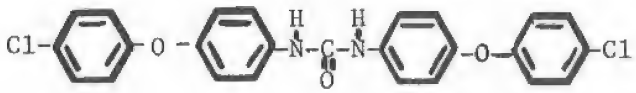
3-(p-Hydroxyphenyl)-1-methoxy-1-methylurea



p-Bromophenylurea

Chloroxuron (Tenoran) [N-(p-Chlorophenoxyphenyl)-N',N'-dimethylurea]

Zea mays, Vicia faba, Phaseolus vulgaris, Galinsoga parviflora, Polygonum convolvulus were exposed to labeled N'-(4-chlorophenoxy)-phenyl-N,N-dimethylurea. After column and thin-layer chromatography, N'-(4-chlorophenoxy)-phenyl-N-methylurea and N'-(4-chlorophenoxy)-phenylurea were identified by infra red. Evolution of labeled CO_2 was observed, indicating metabolism to the p-chlorophenoxy-p'-aminobenzene. Using column chromatography, a compound was eluted on the position of N,N'-bis(4,4'chlorophenoxy)urea (532).



Vamidothion [0,0-Dimethyl S-(N-methylcarbamoylethyl thioethyl) phosphorothioate]

In the urine of mice fed labeled vamidothion, an unknown and the hydrolysis products of vamidothion were observed: mono- and di- methyl phosphate and inorganic phosphate. These materials were also found in the American cockroach and the green rice leafhopper after treatment with vamidothion. Rice and apple leaf contained desmethylvamidothion, dimethyl phosphate and inorganic phosphate (1039).

When vamidothion was incubated with a rat liver homogenate, mono- and di-methyl phosphate were found. A compound believed to be dimethyl phosphorothiolate was also observed. With house fly homogenates, only the mono- and di-methyl phosphates were identified (1039).

VC-13 [0-(2,4-Dichloropheny1)0,0-diethyl phosphorothioate]

Studies have shown that non-hydrolyzed VC-13 residues were greatest in fat and fatty tissues of the body and that free 2,4-dichlorophenol, a hydrolysis product of VC-13, occurred primarily in the kidney and liver. Residues were at a maximum at 7 days after exposure; declined gradually; and were eliminated in 6 weeks by cattle and in 8 weeks by sheep and goats. Cattle were sprayed with, and sheep and goats were dipped in, solutions with the desired concentrations (1532).

Vernolate [S-Propyl dipropylthiocarbamate]

Both peanut (Arachis hyogaea L.) and soybean (Glycine max (L) Merr.) readily absorbed vernolate. The chemical was degraded to CO_2. Two major and two minor metabolites were indicated, but not identified, by paper chromatography (1708).

(See also Tillam.)

Warfarin [3-(α-acetonylbenzyl)-4 hydroxycoumarin]

After incubation of warfarin with subcellular rat liver fractions, 6-, 7-, and 8-hydroxywarfarin were formed. The reaction required molecular oxygen and depended on the presence of an NADPH generating system. One other metabolite was observed but not identified beyond the fact that it was not a sulfate or glucuronide. The predominant metabolite, 7-hydroxy-warfarin, was also found in plasma of rats given warfarin (1701).

Zectran [4-dimethylamino-3,5-xylyl methylcarbamate]

The metabolism of zectran was studied by feeding labeled material to a male beagle dog. Urine was collected and fractionated and metabolites isolated and identified by chromatography. Sulfates and glucuronides were present in the urine as water soluble conjugates of 4-dimethylamino-3,5-xylenol and 2,6-dimethylhydroquinone. Three other materials were not identified (1503, 1504).

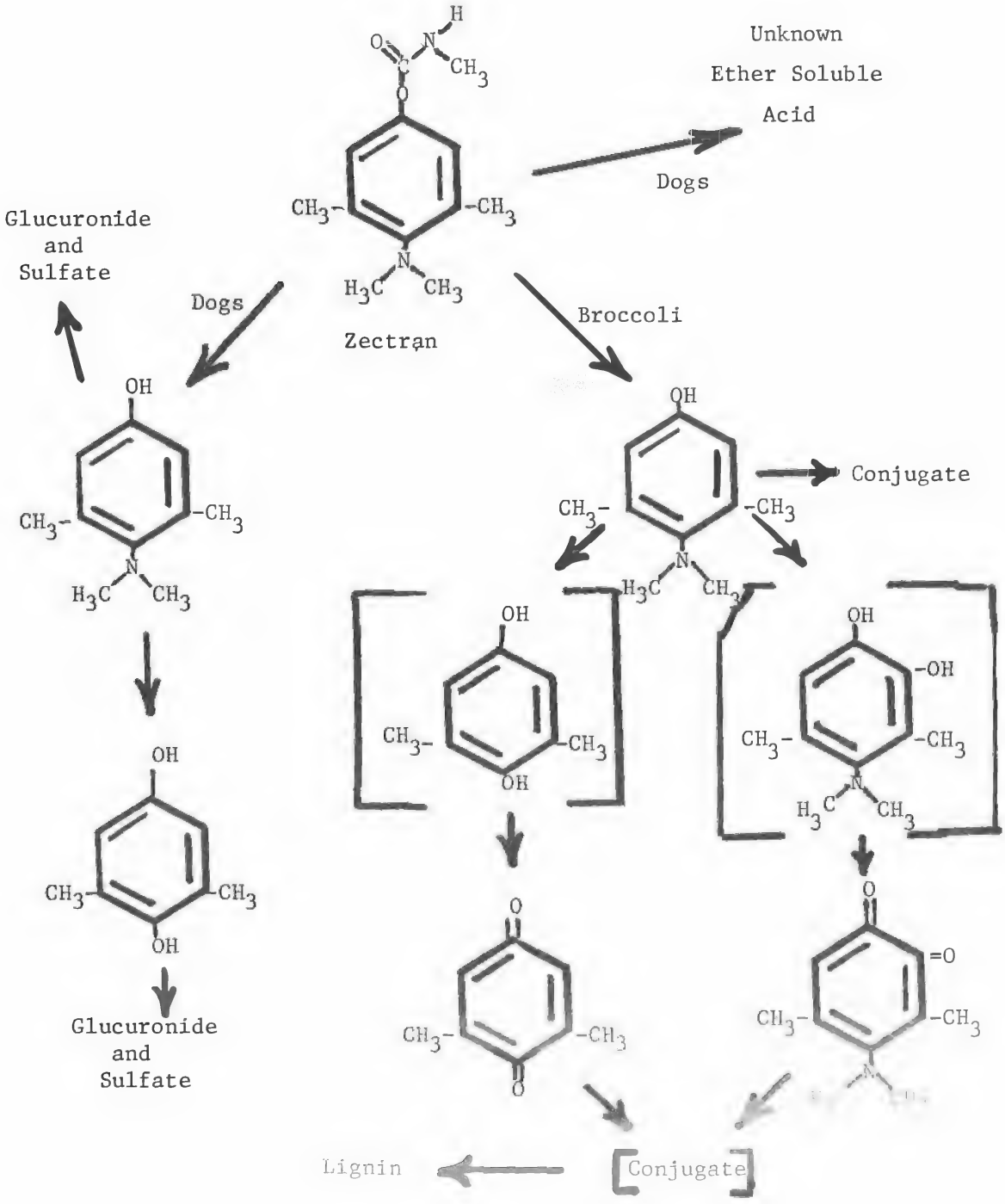
After incubation of zectran with rat liver microsomes and $NADPH_2$, 4-dimethylamino-3,5-xylyl N-hydroxymethylcarbamate and the 4-methylamino-4-amino, and 4-methylformamido-3,5-xylyl methylcarbamates were observed (1110, 1111, 1112). Similar results were obtained with fly homogenates. In addition to the four metabolites indicated, N-hydroxy zectran was also observed (3).

When applied to broccoli, in addition to 4-dimethylamino-3,5-xylenol and 2,6-dimethylhydroquinone, 2,6-dimethyl-p-benzoquinone and 4-dimethylamino-3,5-dimethyl-o-benzoquinone were also found. Examination of methanol insoluble material indicated incorporation of zectran into the lignin where it probably is permanently stored (1503, 1504).

After treatment of bean plants with zectran, at least eight compounds were found. Of these, 4-formamido-3,5-xylyl methylcarbamate, 4-methylformamido-3,5-xylyl methylcarbamate, 4-methylamino-3,5-xylyl methylcarbamate, 4-amino-3,5-xylyl methylcarbamate were identified (3, 845).

UV irradiation of zectran solutions affects zectran; however, no products have been identified (316, 391). Zectran on TLC plates was exposed to ultraviolet light (2537A) for 20 nours. After developing the chromatogram in one direction, the plate was again exposed to ultraviolet light for 20 hours, followed by development in the second direction and subsequent radioautography. From zectran, at least 12 degradation products were observed. Of these, only four were identified: the methylamino, amino, methylformamido, and formamido derivatives. These same major products were obtained from zectran when it was exposed to sunlight on bean foliage. The minor degradation products were the same in number and chromatographic position when formed on plant foliage or on TLC plates (1, 2).

After exposure to zectran, the larvae of spruce budworm and tobacco budworm converted zectran to nine metabolites; housefly larvae producted ten metabolites. Four of these found in all three insects, were identified: 4-methylamino-, 4-amino-, 4-methylformamido-, and 4-formamido- 3,5-xylyl-methylcarbamates. One other compound was believed to be N-hydroxymethyl zectran (1780).


Unknown
Ether Soluble
Acid
Dogs
Glucuronide
and
Sulfate
Dogs
Zectran
Broccoli
Conjugate
Glucuronide
and
Sulfate
Lignin
[Conjugate]

Zinophos [0,0-Diethyl O-2-pyrazinyl phosphorothioate]

The persistence of zinophos in soil was studied in the laboratory and in the field. A curvilinear regression equation was derived that could be applied satisfactorily to the degradation curves (544).

$$y = b_o + b_1 x + b_2 x^{1/2}$$

After soil was treated with zinophos, the hydrolysis product 2-pyrazinol was detected. The oxygen analog of zinophos was unstable in soil and was not detected. When C^{14}-ring-labeled zinophos was used, $C^{14}O_2$ was also observed (542). Information available indicated that the hydrolytic product 2-pyrazinol was rapidly metabolized to smaller fragments (787).

Zytron [O-(2,4-Dichlorophenyl) O-methyl N-isopropylphosphoramidothioate]

Aspergillus clavatus degraded zytron. Small amounts of 2,4-dichlorophenol were detected but the latter did not accumulate (451).

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APPENDIX 1

PHYSICAL CONSTANTS OF SOME ORGANOPHOSPHATE COMPOUNDS*

$$Kt = \ln \frac{C_o}{C}$$ Where C_o = Initial concentration and
C = Concentration at time "t."

This then reduces to the following for the half-life:

$$t_{50} = \frac{1}{K} \ln 2$$

or

$$t_{50} = \frac{0.693}{K}$$

Because of the low solubility of preparations of the "E-605" group, the experiments in these cases were conducted in 20% aqueous methanol at 70°C. At 40°C some of the substances give precipitates, so that the measurements were uncertain.

COMPOUND	Half-Life 70° [HRS.]
E-605 (IV)	64
Methyl-E-605 (XVIII)	13
Isochlorthion (XX)	9
Chlorothion (XIX)	7.3
PREP. S 133 (XXI)	5.5

*(1049)

Table I

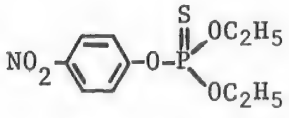
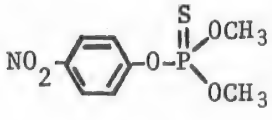
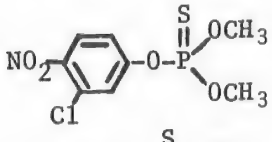
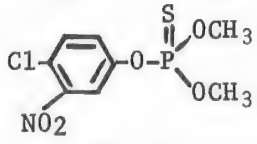
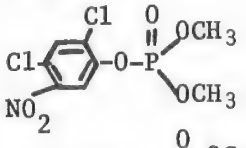
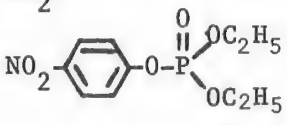
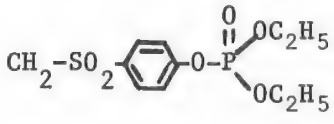
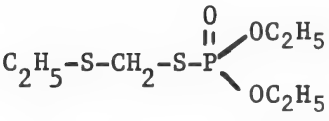
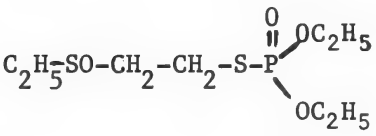
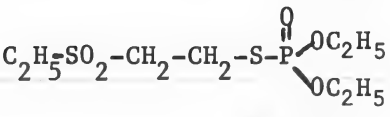
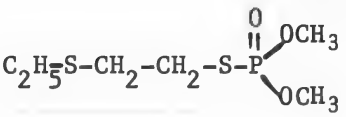
COMPOUND	FORMULA	M.P. [°C]	B.P. [mm]	B.P. [°C]
1. E 605 Series				
E 605 (IV)		6	0.1	122
Methyl-E 605 (XVIII)		35	0.1	119
Chlorthion (XIX)		21	0.1	125
Isochlorthion (XX)		-	0.1	118
S 133 (XXI)		82	0.2	132
E 600 (I)		-	0.1	118
Prep. S 776 (XXII)		-	-	-
2. Systox Series				
PO-Systox (VII)		-	0.8	120
PO-Systox-sulfoxide (XXIII)		-	-	-
PO-Systox-sulfone (XVII)		-	0.15	144
PO-Methylsystox (VIII)		-	0.7	112

Table I (continued)

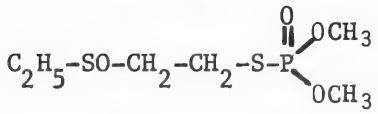
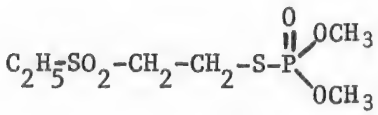
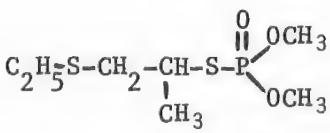
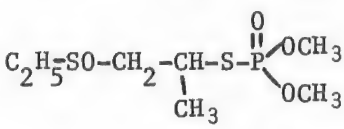
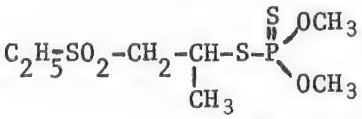
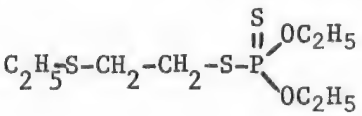
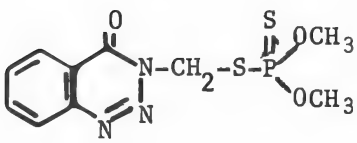
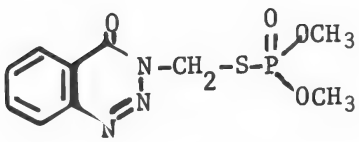
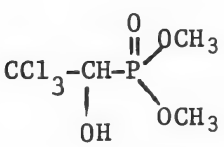
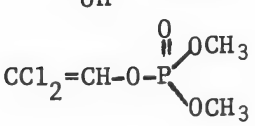
COMPOUND	FORMULA	M.P. [°C]	B.P. [mm]	B.P. [°C]
PO-Methylsystox-sulfoxide (XXIV)		-	0.01	106
PO-Methylsystox-sulfone (XXV)		60	0.12	144
Prep. S 481 (XXVI)		-	0.01	83
Prep. S 410 (XXVII)		-	-	-
Prep. S 404 (XXVIII)		-	0.01	114
Disyston (XI)		-	0.2	103
3. Guthion Series				
Guthion (IX)		74	-	-
PO-Guthion (XXIX)		83	-	-
4. Dipterex Series				
Dipterex (XIII)		83	0.1	100
DDVP (II)		-	1	74

Table II. The hydrolysis constant is in K-100(h^{-1}).
In parenthesis, the half-life in days. All measurements were made at pH 1-5 where the compounds exhibit greatest stability. At this pH the solution had minimal effects on hydrolysis.

COMPOUND	0°	10°	20°	30°	40°	50°	60°	70°	$\frac{K30°}{K20°}$
E 600 (I)	0.0054	0.0024	0.009	0.031	0.10	0.30	0.90	2.4	3.45
	(5350)	(1200)	(320)	(93)	(29)	(9.6)	(3.2)	(1.2)	
E 605 (IV)	0.00021	0.00096	0.0042	0.016	0.058	0.19	0.61	1.76	3.80
	(13800)	(3000)	(690)	(180)	(50)	(15)	(4.75)	(1.65)	
Prep. S 776 (XXII)	0.0054	0.0024	0.009	0.031	0.10	0.30	0.90	2.4	3.45
	(5350)	(1200)	(320)	(93)	(29)	(9.6)	(3.2)	(1.2)	
PO-Systox (VII)	0.0005	0.003	0.014	0.054	0.185	0.64	2.24	5.91	3.85
	(5800)	(960)	(206)	(54)	(15.6)	(4.5)	(1.29)	(0.49)	
PO-Systox-sulfoxide	0.00018	0.00085	0.0037	0.014	0.051	0.16	0.58	1.48	3.80
(XXIII)	(16000)	(3400)	(780)	(206)	(57)	(18)	(5.0)	(1.95)	
PO-Systox-sulfone (XVII)	0.00018	0.00085	0.0037	0.014	0.050	0.16	0.55	1.49	3.80
	(16000)	(3400)	(780)	(206)	(57)	(18)	(5.0)	(1.94)	
PO-Methylsystox (VIII)	0.002	0.008	0.033	0.122	0.385	1.42	4.5	14.2	3.70
	(1450)	(360)	(88)	(25.8)	(7.5)	(2.03)	(0.65)	(0.204)	
PO-Methylsystox-	0.0006	0.003	0.0122	0.047	0.16	0.56	1.75	4.75	3.85
sulfoxide (XXIV)	(4800)	(970)	(236)	(61.7)	(18)	(5.2)	(1.65)	(0.61)	
PO-Methylsystox-sulfone	0.0006	0.003	0.012	0.047	0.16	0.56	2.17	4.75	3.85
(XXV)	(4800)	(970)	(236)	(61.7)	(17)	(5.2)	(1.33)	(0.61	
Prep. S 481 (XXVI)	0.2	0.6	1.60	4.08	9.86				2.55
	(14.5)	(4.8	(1.8)	(0.71)	(0.293)				
Prep. S 410 (XXVII)	0.00055	0.0027	0.012	0.045	0.162	0.53	1.53	3.94	3.75
	(5200)	(1070)	(240)	(63)	(18)	(5.5)	(1.9)		

Table II. The hydrolysis constant is in K-100(h^{-1}).
In parenthesis, the half-life in days. All measurements were made at pH 1-5 where the compounds exhibit greatest stability. At this pH the solution had minimal effects on hydrolysis (continued)

COMPOUND	0°	10°	20°	30°	40°	50°	60°	70°	$\frac{K30°}{K20°}$
Prep. S 404 (XXVIII)	0.00055 (5200)	0.0027 (1070)	0.0125 (240)	0.047 (63)	0.170 (18)	0.53 (5.5)	1.53 (1.9)	4.91 (0.59)	3.75
Disyston (XI)	0.00012 (23200)	0.0006 (4830)	0.0026 (1110)	0.010 (290)	0.037 (78)	0.12 (24)	0.37 (7.8)	1.06 (2.7)	3.85
Guthion (IX)	0.00055 (5200)	0.0027 (1070)	0.012 (240)	0.047 (61.5)	0.16 (18)	0.53 (5.46)	1.53 (1.9)	4.75 (0.61)	3.75
PO-Guthion (XXIX)	0.002 (1450)	0.008 (360)	0.035 (82.5)	0.13 (22.2)	0.45 (6.4)	1.45 (2.0)	4.93 (0.585)	13.8 (0.21)	3.7
Dipterex (XIII)	0.00025 (11600)	0.0012 (2400)	0.0055 (526)	0.0207 (140)	0.07 (41)	0.27 (10.7)	0.90 (3.2)	2.56 (1.13)	3.75
DDVP (II)	0.0028 (1030)	0.012 (240)	0.047 (61.5)	0.167 (17.3)	0.503 (5.8)	1.745 (1.66)	5.03 (0.58)	17.6 (0. 64)	3.60
Chlorthion (XIX)	0.001 (2900)	0.0048 (600)	0.021 (138)	0.08 (36)	0.286 (10)	0.95 (3.0)	2.93 (1)	8.34 (0.35)	3.8
Methyl-E 605 (XVIII)	0.0008 (3600)	0.0038 (760)	0.0165 (175)	0.064 (45)	0.230 (12.5)	0.72 (4.0)	2.16 (1.34	6.12 (0.47)	3.85
Thimet (XII)	0.014 (200)	0.08 (36)	0.4 (7.2)	1.83 (1.6)	7.14 (0.4)	- -	- -	- -	4.6

Table III. Hydrolysis constant is in $10^{-2}K(h^{-1})$ at 70°C.
In parentheses, the half-life in hours.

COMPOUND	n-HCl	pH=1	2	3	4	5	6	7	8	9
E 600 (I)	7.36	3.77	3.06	3.00	2.85	2.85	3.87	6.0	7.5	33.5
	(9.4)	(18.5)	(22.7)	(23)	(24.4)	(24.4)	(18)	(11.5)	(9.2)	(2.1)
E 605 (IV)	1.84	2.05	2.55	3.28	3.97	3.56	5.36	8.86	16.7	25.2
	(37.7)	(34)	(27)	(21)	(17.5)	(19.5)	(13)	(7.8)	(4.1)	(2.7)
Prep. S 776	8.38	3.56	3.11	3.06	3.36	3.56	4.48	8.0	8.17	25.6
(XXII)	(8.3)	(19.5)	(22.3)	(22.6)	(20.6)	(19.5)	(15.5)	(8.7)	(8.5)	(2.7)
PO-Systox (VII)	0.6	2.49	5.69	6.79	6.85	7.16	7.30	8.13	8.18	16.3
	(115)	(28)	(12)	(10)	(10)	(10)	(9.5)	(8.5)	(8.5)	(4.2)
PO-Systox-sulfoxide	2.77	1.70	1.72	1.86	1.94	2.06	2.20	3.50	7.42	42.2
(XXIII)	(25)	(41)	(40)	(37)	(36)	(34)	(32)	(20)	(9.3)	(1.6)
PO-Systox-sulfone	3.31	1.91	1.80	1.84	1.85	2.25	6.51	69.5	188	-
(XVII)	(20)	(36)	(39)	(38)	(38)	(31)	(10.6)	(1)	(0.37)	-
PO-Methylsystox	8.7	13.3	13.7	14.2	14.1	16.2	18.2	19.8	20.3	55.5
(VIII)	(8)	(5.2)	(5)	(4.9)	(4.9)	(4.3)	(3.8)	(3.5)	(3.4)	(1.25)
PO-Methylsystox-	13.5	5.6	5.7	5.6	5.7	4.5	5.6	11.0	26.1	140.5
sulfoxide (XXIV)	(5.1)	(12.4	(12.2)	(12.4)	(12.2)	(15.4)	(12.4)	(6.3)	(2.65)	(0.5)
PO-Methylsystox-	20.1	7.35	6.45	6.45	7.4	6.45	13.6	72.3	162	-
sulfone (XXV)	(3.4)	(9.5)	(10.7)	(10.8)	(9.4)	(10.7)	(5.1)	(0.96	(0.42)	-
Prep. S 481	-	132	156	155	158	234	224	246	-	-
(XXVI)	-	(0.52)	(0.44)	(0.45)	(0.44)	(0.3)	(0.31)	(0.28)	-	-
Prep. S 410	15.5	6.83	6.72	6.93	7.25	6.60	7.04	11.9	23.1	138.5
(XXVII)	(4.5)	(10.0)	(10.3)	(10.0)	(9.5)	(10.5)	(9.8)	(5.8)	(3.0	(0.5)
Prep. S 404	18.4	6.93	6.51	6.51	7.25	6.09	8.12	25.0	77.6	-
(XXVIII)	(3.8)	(10.0)	(10.6)	(10.6)	(9.5)	(11.4)	(8.5)	(2.8)	(0.9)	-

Table III. Hydrolysis constant is in $10^{-2}K(h^{-1})$ at 70°C.
In parentheses, the half-life in hours (continued)

COMPOUND	n-HCl	pH=1	2	3	4	5	6	7	8	9
Disyston (XI)	2.88 (24)	1.11 (62)	1.11 (62)	1.10 (62)	1.11 (62)	1.16 (60)	1.56 (44)	2.50 (27.6)	3.22 (21.5)	9.61 (7.2)
Guthion (IX)	8.95 (7.75)	2.92 (24)	5.11 (13.5)	7.72 (9)	9.68 (7.2)	7.80 (8.9)	9.26 (7.5)	14.5 (4.8)	29.3 (2.4)	117 (0.6)
PO-Guthion (XXIX)	30.5 (2.27)	15.4 (4.5)	15.2 (4.5)	15.4 (4.5)	17.3 (4.0)	15.9 (4.4)	17.4 (4.0)	32.8 (2.1)	76.0 (0.88)	- -
Dipterex (XIII)	6.2 (11.2)	2.15 (32)	2.02 (34)	2.09 (33)	2.60 (26.6)	4.54 (15.3)	22.8 (3.0)	100 (0.7)	111 (0.6)	693 (0.1)
DDVP (II)	111 (0.6)	29.6 (2.3)	20.5 (3.4)	20.3 (3.4)	22.9 (3.0)	24.8 (2.8)	48.7 (1.4)	152 (0.45	- -	- -
Chlorthion (XIX)	0.035 (20)	0.06 (11.6)	0.08 (8.7)	0.10 (6.9)	0.11 (6.1)	0.13 (5.3)	0.15 (4.6)	0.19 (3.6)	0.48 (1.4)	- -
Methyl-E 605 (XVIII)	0.022 (31)	0.045 (15.4)	0.058 (12.0)	0.062 (11.2)	0.063 (11.0)	0.065 (10.7)	0.069 (10.0)	0.10 (6.9)	0.26 (2.7)	0.45 1.5

APPENDIX 2

CALCULATIONS FOR
ADSORPTION AND LEACHING OF HERBICIDES

Adsorption

Adsorption of a compound can be measured in a system of fixed soil-solution ratio and varying herbicide concentration. This may be plotted in terms of the empirical isothermal Freundlich relationship $\frac{x}{m} = KC^n$ on a log x log scale. The slope of the line then represents "n". From plots of this type, the total amount of herbicide to be applied may be calculated. Calculations show that the concentration of herbicide to be applied, expressed in kg/ha, is roughly equal to 1/10 the soil concentration $\frac{x}{m}$ at the 1 ppm level. This rule of thumb applies to soils having a high moisture level (531, 532).

$$\frac{x}{m} = KC^n \qquad \frac{kg}{ha} = \frac{x}{10m}$$

$\frac{x}{m} = \frac{\gamma \text{ chemical}}{g \text{ soil}}$ = concentration of chemical fixed on soil

$C = \frac{\gamma \text{ chemical}}{ml \text{ water}}$ = concentration of chemical in solution

K = constant

n = slope

The rate diffusion of herbicides may be calculated from Fick's diffusion equation:

$$m = D \cdot q \frac{C_o - C'}{\delta} t$$

m = mass of herbicide

D = diffusion coefficient

q = surface of herbicide

C_o= solubility of herbicide in water

C'= final concentration of dissolved herbicide

δ = thickness of diffusion layer

t = time in seconds

The reader is also referred to a discussion of "Leaching of Pesticides from Granular Formulations" by Furmidge, Hill and Osgerby (513).

SERIAL
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